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CONTENTS OF PREVIOUS NUMBERS

OCTOBER, 1923. NUMBER 4

Neurological Surgeons: with the Report of One Case. Harvey Cushing, M.D., Boston.

Studies in Decerebration. I. A Method of Decerebration. Lewis J. Pollock, M.D., and Loyal E. Davis, M.D., M.S., Chicago.

Researches on the Pupillary Reactions in Epidemic Encephalitis. Henry G. Mehrtens, M.D., and Otto Barkan, M.D., San Francisco.

Fulminating Encephalomyelitis. Henry Edmund Meleney, M.D., Peking, China.

Spinal Subarachnoid Block; Its Significance as a Diagnostic Sign. Analysis of Fifty-Three Cases. James B. Ayer, M.D., Boston.

Dr. George M. Beard; a Sketch of His Life and Character, with Some Personal Reminiscences. Charles L. Dana, M.D., New York.

Folie à Trois. F. J. Gerty, M.D., and George W. Hall, M.D., Chicago.

Critical Reviews: The Pathology of Motor Paralysis by Lead. George H. Hyslop, A.M., M.D., and Walter M. Kraus, A.M., M.D., New York.

Abstracts from Current Literature:
Malignant Tumors of the Nervous System.—Migraine in Children.—Action of Ether on the Circulation in Traumatic Shock.—Tumor of the Infundibulum, Integrity of the Hypophysis, Adipose Syndrome.—Endocrine Imbalance and Mental Disease.—Sequelae of Epidemic Encephalitis.—War and Acute Delirious Hyperthymia.—Wassermann Reaction and Colloidal Methods in the Spinal Fluid of Neurosyphilities.—The Radiologic Signs of Vertebral Cancer.—Experiments on the Transplantation of the Spinal Cord in Amblystoma. — Familial Myopathy of the Peroneal or Distal Type.—Mental Disorders of Children.—Zona.—Acute Hemorrhagic Encephalitis. — The Rôle of the Labyrinth in Exophthalmic Goiter.—Contribution to the Study of Apraxia.—Significance of the Term Hippocampus.—Dysthymic Children.—Syndromes of the Infundibular Hypophysial Group in Epidemic Encephalitis.—Lower Half Headache of Nasal Origin.—Cell Structure of the Superior Olive in Man.

Society Transactions:
Philadelphia Neurological Society.

Book Reviews.

NOVEMBER, 1923. NUMBER 5

Central Pain in Syringomyelia and Dysesthesia and Overreaction to Sensory Stimuli in Lesions Below the Optic Thalamus. William G. Spiller, M.D., Philadelphia.

Central Pain from Lesions of the Pons. T. H. Weisenburg, M.D., and S. S. Stack, M.D., Philadelphia.

Spastic Paraplegia in Flexion. A Case Due to a Meningeal Tumor Compressing the Lower Cervical Cord on the Anterior and Right Lateral Aspects. Walter F. Schaller, M.D., and Philip K. Gilman, M.D., San Francisco.

A Study of Bladder and Rectal Disturbances in Spinal Cord Tumors. Byron Stookey, A.M., M.D., New York.

Visual Hallucinations as a Cerebral Localizing Phenomenon with Especial Reference to Their Occurrence in Tumors of the Temporal Lobes. Gilbert Horrax, M.D., Boston.

Gastric Secretory Functions in the Psychoses. Clifford B. Farr, M.D., and Charles W. Lueders, M.D., Philadelphia.

News and Comment:
Parkinson's Shaking Palsy.

Abstracts from Current Literature:
Diseases of the Striate System.—The Etiology and Pathology of Sciatica.—The Roentgen-Ray Treatment of Toxic Goiter.—Dystrophia Myotonica.—The Significance of the Frontal Lobes for the Higher Psychic Functions.—Brain Tumors in Young Children.—The Phenomenon of the Index Finger.—Psychic Disturbances and Hypophysial Tumors. — Raynaud's Disease.—Epidemic Encephalitis and Herpes Febrilis.—Huntington's Chorea and Migraine.—Influenzal Meningitis.—Case of Epilepsy of Twenty-Two Years' Standing Due to Calcified Endothelioma or Perithelioma.—Serous Meningitis and Cerebral Tumor.—Experiments in Treating Epidemic Encephalitis with Casein Intraspinally.—Subdivisions of the Neural Folds in Man.—The Cerebrospinal Fluid in Postdiphtheric Paralysis.—Venous Angioma of the Cerebral Cortex.—Studies of the Cerebrospinal Fluid with an Acetic Anhydride Sulphuric Acid Test.—A Case of General Paralysis Having an Evolution of Seventeen Years.—Neuropathic Manifestations in Infants and Children as a Result of Anaphylactic Reaction to Foods.—Trophic Changes in Syringomyelia.—Cell Structure of the Superior Olive in Man.—Chorea Cruciata.—The Introduction of Antimeningococcus Serum by Cistern Puncture.

Society Transactions:
American Neurological Association.

Book Reviews.

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Archives of Neurology and Psychiatry

Vol 10 DECEMBER, 1923 No. 6

NOTES ON A SERIES OF INTRACRANIAL TUMORS AND CONDITIONS SIMULATING THEM

TUMOR SUSPECTS; TUMORS UNVERIFIED; TUMORS VERIFIED *

HARVEY CUSHING, M.D.
BOSTON

This discussion may be opened by calling attention to Table 1 as a point of departure for the several matters I shall briefly touch on in connection with the patients with proved or presumptive tumors at present in the hospital wards. It gives in summary a working subdivision of the cases in our tumor series up to May 1 of this year.

TABLE 1.—*Cases in the Tumor Series*

	Number	Percentage	Total
Tumors histologically verified			868
Gliomas (so diagnosed) of all types	362	41.7	
Adenomas (chiefly pituitary)	169	19.5	
Meningiomas ("dural endotheliomas")	99	11.4	
Neurinomas (acoustic)	77	8.9	
Congenital tumors	54	6.2	
Craniopharyngeal pouch cysts	42		
Cholesteatomas and dermoids	8		
Teratomas	4		
Granulomatous tumors	36	4.2	
Tuberculomas	23		
Syphilomas	13		
Metastatic and invasive tumors (varia)	34	3.9	
Papillomas (choroid-plexus tumors)	13	1.5	
Angiomas	8	0.9	
Miscellaneous and unclassified	16	1.8	
Tumors unverified but presence unquestioned			476
Tumor suspects: probably not tumor, or so proved			314
Total listed cases			1,658

A special card catalogue of these cases is kept up to date. Naturally the cards may subsequently be shifted from time to time as diagnoses are corrected or new information is secured. The material they represent serves as the basis of our special tumor studies.

* The basis of a clinic before the American Neurological Association at the Peter Bent Brigham Hospital, Boston, May 31, 1923. Much was left to the audience in the way of recognizing the status of each patient. All that can be indicated here was whether they were walking, bed, or wheelchair cases. Owing to lack of space there are omitted here a number of the original illustrations of patients, tumors and microscopic sections.

We have found it convenient for clinical purposes to file the cards under three main headings: *tumor suspects,* of which there have been recorded 314 examples; *tumors unverified,* 476 cases; and *tumors verified,* which on the first of this month numbered 868 cases. Each of these separate groups needs some elucidation. There are at present thirty-one additional cases in the hospital which will come to be included in one of these three groups. Some have already been operated on; some await operation; some will escape it. In addition, I have asked a few other patients recently discharged to report at this session.

There are certain definite questions we must keep before us in every case. (1) Is tumor or some other lesion responsible for the clinical syndrome? (2) If tumor, what is its location? (3) Its site having been determined, what is its probable character? (4) Is it operable? (5) What is the end result? An answer to the first of these questions may be given in most cases after a thorough neurologic study, even without an exploratory operation, though in some doubtful instances this may be required. The cases which we are enabled to discard by one means or another from the true tumor list we may first of all consider.

TUMOR SUSPECTS

These, as previously emphasized in papers by Dr. Bailey and Dr. Locke, are clinically the most interesting cases of all. They require a general neurologic training if they are to be differentiated from tumor, which indeed they oftentimes cannot be until the presence, or absence, of a growth is made certain by operation or necropsy.

Naturally these patients are sent into the clinic for some outstanding symptom—headache, convulsions, loss of vision, or something of the kind which suggests a possible tumor; and often the best we can do is temporarily to diagnose the case on a symptomatic basis—for example, as "Cephalalgia: cause unknown." But we have been deceived so often by discarding tumor in favor of another diagnosis that we incline toward this diagnosis until it can be disproved, rather than the reverse. We would, for example, look with great suspicion on a patient in whom a choked disk accompanies an encephalitic syndrome, whereas some of you might incline toward encephalitis in view of the general picture.

I cannot give the exact number of cases that have passed through our hands with this "Tumor Suspect" diagnosis, which later on, here or elsewhere, have been proved to be tumor. The partial list [1] (Table 2)

1. The list comprises the "suspects" admitted to the clinic during the past few years since we have been recording these conditions under tumor. With it have been included a few cases from the earlier series, then designated as "pseudo tumor."

includes only those cases which originally entered the clinic as possible tumor, and whose symptoms we must still regard as due in all probability to some other condition.

The list is too long to give in full and these few entries must suffice. The sixty-seven other cases include such varied diagnoses as optic neuritis, diabetic coma, myasthenia gravis, neuralgia, aphasia, and so on.

Even at necropsy one may be deceived. In a recent revision with Dr. Bailey of the diagnoses of my early Baltimore series, we have been able to correct a good many errors. I may give in illustration one case which has gone all these years under the caption of "pseudo tumor," or, as it would now be classified, "Tumor Suspect: Hydrocephalus." The patient was a boy, aged 9, who was nearly blind from an obvious

TABLE 2.—*Tumor Suspects*

A. Symptomatic diagnoses (cause undetermined)

Convulsions (varia)	28	Hemiplegia	4
Amblyopia (varia)	14	Ataxia	4
Neuroses (varia)	13	Psychoses (varia)	3
Cephalalgia (varia)	11	Xanthochromia	1
Dementia (varia)	9	Nystagmus	1
Hysteria (varia)	6	Ophthalmoplegia Externa	1
Hydrocephalus	5		

B. Organic diagnoses (proved or reasonably certain)

Cerebral Vascular Disease	36	Abscess	9
Arachnoiditis	29	Labyrinthine Disease	7
Cerebral Syphilis	15	Aneurysm	6
Meningitis (varia)	13	Nephritic Edema	3
Encephalitis (varia)	12	Cerebellar Apoplexy	2
Sclerosis (varia)	10		

obstructive hydrocephalus, due as we thought to a subtentorial tumor. He died some weeks after a negative cerebellar exploration and at necropsy nothing was found to account for his hydrocephalus which was ascribed to a possible ependymitis or arachnoiditis. Reinvestigation of the tissues with a study in serial sections of the brain stem has disclosed a microscopic subependymal glioma occluding the iter (Fig. 1). Had this unfortunate boy been "discharged unrelieved" the diagnosis of "tumor unverified" would have been recorded—a diagnosis nearer the truth than that of "pseudo tumor" actually made after the necropsy with the subsequent incomplete study of the brain.

The first patient will illustrate what we mean by a "tumor suspect" syndrome.

CASE 1 (Wheel-chair).—This lady is in her seventy-eighth year. She entered the hospital a few weeks ago complaining of facial pain and blindness of the right eye. There is marked internal squint of this eye; the pupil gives no reaction

to light, and the nerve head shows complete primary atrophy. In addition to this, there is anesthesia of the entire trigeminal skin field on the right side. She has no other neurologic symptoms.

There can be no question as to the site of the lesion, which involves the right optic nerve, the abducens, and the sensory portions of the trigeminus; but the nature of such a lesion without other facts, particularly those gained from her past history, might be open to considerable doubt. My first impression was that she might have a meningioma arising from the right trigeminal envelopes.

She recalls very imperfectly the chronology of her symptoms. Her account is that she enjoyed good health until five years ago, when, after exposure to a cold wind, she had a sudden attack of pain in the right eye. This was regarded as neuralgia, for which she subsequently had one turbinate removed. One of

Fig. 1.—Section of iter showing subependymal glioma. Complete occlusion existed at another level. × 30.

her former attendants, however, reports that she consulted him thirteen years ago, when she had partial ptosis of the right eye, with trigeminal pain and a turning-in of the eye. These symptoms must have largely subsided, for seven years later she consulted another physician because of temporary paresis of the right external rectus, and he noted that she had marked arteriosclerosis.

Three years ago she sustained a fracture of the hip from which she has made a fairly good functional recovery. At about this time she thinks her present strabismus began, with gradual loss of vision in the eye.

For a year there has been numbness of the right side of the face, increasing in extent and degree. It is not clear in which order the trigeminal divisions became involved, but at present there is practically total anesthesia over all three of them and the corneal reflex is absent. There have been no uncinate symptoms.

In spite of her original imperfect history, my assistant Dr. McKenzie jumped at the correct diagnosis, and his opinion was corroborated by the roentgenograms. They show a crescentic shadow in the right temporal fossa alongside the sella, unquestionably due to calcification in the wall of a cyst. We have seen something similar, to be sure, in a patient with a dermoid in this region—a case recently reported by Dr. Horrax.[2] However, in view of her arteriosclerosis it is much more likely that the calcification lies in the wall of an aneurysm; and as she is to be discharged untreated, the diagnosis will read, "Brain tumor suspect: aneurysm of internal carotid: unverified."

We have been on the alert for these aneurysms during the past three or four years, ever since Dr. Symonds of Guy's Hospital while here made a correct diagnosis during life on a patient who had entered the hospital *in extremis* and who showed unilateral ptosis, subretinal hemorrhages, and bloody cerebrospinal fluid.[3] The experience led to a change in the presumptive diagnosis on the cards of several cases in the list of "tumor suspects"—cases which had previously been recorded as unilateral ophthalmoplegia, ophthalmoplegic migraine, pachymengitis hemorrhagica, and basilar syphilis.

To be sure, the aneurysm in this patient, if aneurysm it actually be, has never ruptured. Moreover, it is larger by far than the usual mycotic aneurysms of this region, which are small, pea-sized affairs and usually give no symptoms till they have ruptured (Fig. 2). The first symptoms often resemble a stroke of apoplexy with bleeding into the meninges; and, under these circumstances, if a fatality ensues the small lesion, even should its presence have been suspected, may easily escape notice at necropsy unless great care is exercised. Curiously enough, recovery is not uncommon even when rupture has occurred. There may indeed be recurrent hemorrhages.

I see nothing surgical about this case. However, the symptoms which the patient shows, essentially those of a stationary tumefaction in the middle cerebral fossa, might easily lead the unwary into a useless and dangerous exploration.

I will show you another example of tumor suspect—one which has been proved not to be tumor only after exploration.

Case 2 (Walking).—This man, 47 years of age, was recommended to the hospital a month ago by Dr. Beverly Tucker. His chief complaints were loss of vision, diplopia, and defective memory. For about six years, following what seems to have been either the grippe, encephalitis, or acute articular rheumatism followed by endocarditis, he has been emotionally unstable. He finally gave

2. Horrax, Gilbert: A Consideration of the Dermal Versus the Ependymal Cholesteatomas Having Their Attachment in the Cerebral Envelopes, Arch. Neurol. & Psychiat. **8**:265 (Sept.) 1922.

3. This, with other samples of intracranial aneurysm of the internal carotid which have been diagnosed during life, has been reported by Dr. C. P. Symonds in Guy's Hospital Reports for April, 1923, p. 139.

up his profession and took to farming. During 1919 and the following year he occasionally had transient diplopia lasting three or four days. In March, 1921, he had a general convulsion and the next year several more.

Physical examination was essentially negative. What in our opinion was taken by our predecessors for an optic atrophy is the pallor due to an unusually large lamina cribrosa in each eye. Moreover, we were unable to substantiate the supposed homonymous field defect they had observed. He showed merely marked memory defect, emotional instability and lack of initiative. He was incooperative and suffered from insomnia. There was considerable tremor of the outstretched hands, and the abdominal reflexes were inactive. Roentgenograms

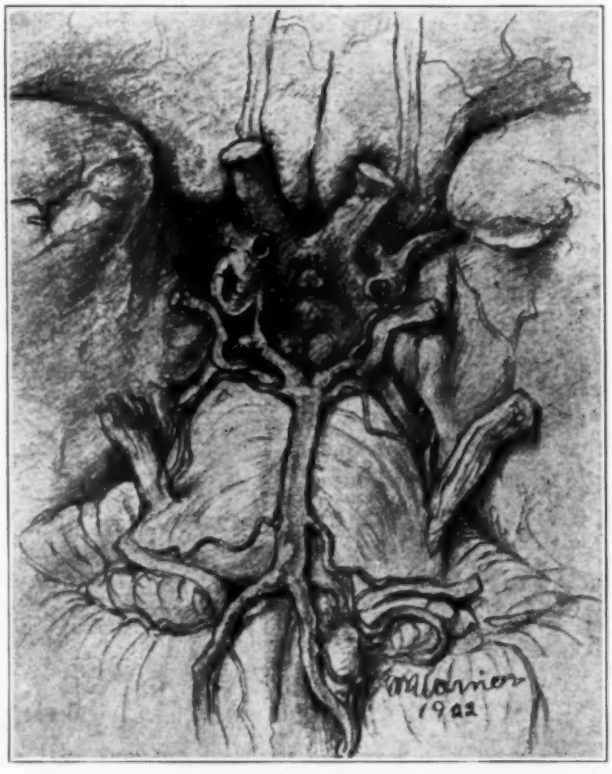

Fig. 2.—Sketch of the ruptured aneurysm in Dr. Symond's report.

disclosed a calcified plaque of the anterior falx (Fig. 3). Repeated Wassermann tests and detailed studies of the cerebrospinal fluid including the colloidal gold curve had been negative.

Our tentative diagnosis was "presenile dementia" of some sort; but as there had been a difference of opinion it was thought that light might be shed on the matter by pneumoventriculograms. These were taken, but unfortunately proved misleading. The left ventricle was normal in size and contour. The right was much smaller than the left and showed irregularity and imperfect filling. No lateral dislocation was apparent. There was no way definitely to clear up the diagnosis without an exploration. I rather expected to find a right frontal endothelioma arising from the falx.

Operation disclosed a markedly atrophic frontal lobe with many empty thread-like vessels and thickening of the leptomeninges. A fragment, including one of the empty cortical vessels, was excised for examination. This shows merely an extreme degree of gliosis, no abnormalities being present in the walls of the empty arteriole—certainly nothing suggesting syphilitic vascular disease.

The operation has had an unexpectedly beneficial effect. His entire mental outlook is changed. He has begun reading again; takes a renewed interest in his surroundings; sleeps well; has been attending clinics and thinks of resuming his profession.

Fig. 3 (Case 2).—Anteroposterior roentgenogram showing calcified placque on anterior falx.

There is no better time to register helpful psychotherapeutic impressions than after an operation. Because patients often appear, however, to be subjectively benefited by them rarely justifies their performance. One may be very easily misled and ascribe too much to the operation itself. It is this sort of self deception, in which surgeon and patient and relatives often share, that has led to innumerable futile operations for old birth palsies and hopeless conditions of a similar kind. This man's

operation was purely exploratory. It is of course gratifying that he is subjectively improved, but this can hardly be directly ascribed to the operation. He is about to be discharged and the case will be recorded as "Brain tumor suspect: cerebral sclerosis: verified."

The next three patients—all three of them with threatened loss of vision—are further examples of suspected tumor.

Case 3 (Walking).—Mr. S., aged 31, an active business man inclined to work under high tension, first entered the hospital in January of this year on the recommendation of Dr. J. W. Courtney with the diagnosis of a "tumor in the lower lip of the left calcarine fissure." This was a little fine, but unquestionably he has a lesion of some sort involving the left visual pathway, for he shows an homonymous field defect involving the right upper quadrants.

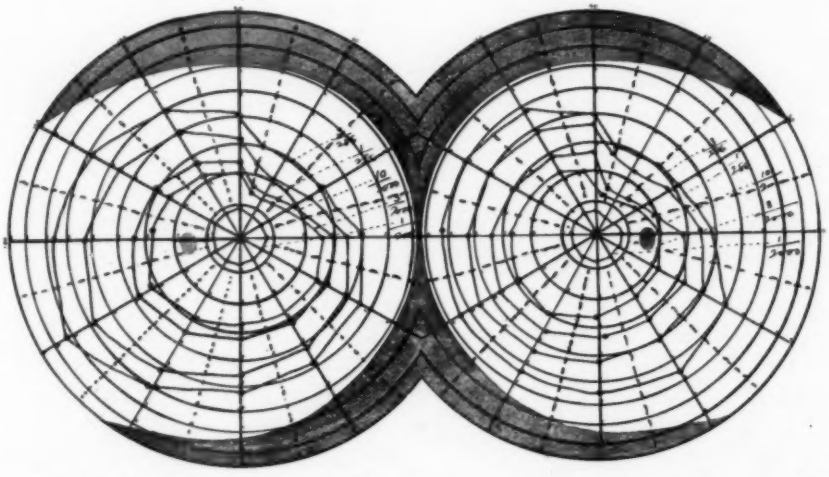

Fig. 4 (Case 3).—Persistent right upper homonymous field defects. V.O.S. 20/30; V.O.D. 20/30.

Obscuration of vision with some mild headaches represent his chief complaint, but the field defect is of the type we are more inclined to associate with a temporal than with an occipital lesion.

The onset of symptoms was rather abrupt for tumor, being more suggestive of a vascular lesion. Without loss of consciousness he experienced a sinking feeling with transient loss of power in his right side some time last August; and, soon after, he himself noticed the constriction in the right upper visual fields.

Since then he has never felt quite himself though he has continued with his business affairs. There have been headaches, night sweats, and "muscular quaverings" in his right side. He also gave a history of vague abdominal sensation ("sinking spells") accompanied by gustatory impressions suggestive of uncinate seizures.

Physical and neurologic examinations revealed nothing except the homonymous field defect (Fig. 4) and a slightly edematous papilla in the left eye with

marginal obscuration of the disk. Feeling that delay was justified, he was discharged untreated, with a diagnosis of "tumor unverified: left temporal lobe (?)."

He reentered the hospital two weeks ago. During the four months' interval he had had a good deal of dull frontal headache and felt that his vision was becoming progressively impaired. The perimeter, however, did not corroborate this. The papilledema of the left eye showed no advance.

Ten days ago a subtemporal decompression was performed. There was an excess of fluid, but the temporal lobe appeared quite normal. The area is now quite flat though it bulges slightly at times when he tends to feel a headache—a symptom, I am glad to say, from which he has been comparatively free since his operation.

What is this? An arachnoiditis, a vascular lesion, or tumor? I think we will now have to recede from our presumptive tumor diagnosis in favor of the less definite "tumor suspect." In any event, we need no longer fear that we are neglecting the possible secondary pressure effects of tumor, and can now await further developments, if there are to be any, with some assurance that his cephalalgia meanwhile will be relieved and that his optic nerves will suffer no further damage.

What may happen if we wait too long is illustrated by the next two patients. Both entered the hospital the same afternoon.

CASE 4 (Walking, but led).—This woman, 37 years of age, began three years ago to have temporary failure of vision. Two years ago she was given a thorough study in the Neurological Institute where I judge that the diagnosis lay between tumor and retrobulbar neuritis. She had repeated Wassermann tests, rhinologic and roentgen-ray examinations of her sinuses, and so on: all were negative.

During the succeeding two years this low grade of choked disk has persisted, until now she has advanced secondary atrophy with blindness of the right eye and almost complete loss of vision in the left. She has had from the outset some moderate intracranial discomforts, chiefly described as aching in the back of the neck. The neurologic examination, aside from the changes in the optic nerves, is practically negative.

Her chronologic twin, Mrs. LaB., I need not produce. She has had symptoms for only a year, with low grade of choked disk (first observed in February of 1922), and some moderate headaches. For the past month there has been rapid failure of vision with complete blindness on the right side and marked constriction of fields on the left. Aside from inconspicuous nystagmus and choked disk of about 2 diopters, her neurologic examinations have been as completely negative as in the patient who has just been here. She however has the additional factor of high blood pressure—over 200. Cases of this kind are what make up the group on our tumor-suspect cards listed symptomatically as "Optic neuritis: cause undetermined."

I fear that unless a decompression is performed, this patient's vision after another few months will be as irrevocably lost as in the case of the

unfortunate patient who has just gone out. I say this with all deference to Dr. Bernard Sachs who gave us last year a very gloomy picture of his personal experience with decompression operations.[4] He was doubtless referring to cases of unquestioned tumor, but even in the tumor cases, as we shall see later, the measure may be beneficial: indeed, may be our only therapeutic recourse if we are to preserve vision.

We now come to a more definite example of what for want of a better term we classify as "Brain tumor suspect: chronic arachnoiditis." In these cases probably more than any others we are likely to be deceived. The preceding two cases may possibly belong in this same category.

Case 6 (Walking).—This healthy-appearing man, aged 38, is as good an example of our difficulties as any other. This is his second reentry. He came here first in December, 1922, complaining of annoying tinnitus in the right ear, with progressive impairment of hearing. For eighteen months he had been having recurrent attacks of nausea and vertigo accompanied by right-to-left movement of objects. In July, 1922, a diagnosis had been made of toxic vertigo: his tonsils were removed and he was given phenobarbital. Latterly, his attacks had increased in number, often two or three a day, in spite of large doses of bromids. He had become persistently unsteady on his feet.

Neurologic examination revealed incomplete right nerve deafness, moderate nystagmus, positive Romberg sign and some clumsiness in gait. There was some obscuration of the nasal margins of the disks on each side without measurable elevation. The blood Wassermann reaction was negative. Lumbar puncture was not performed.

I inclined to a presumptive diagnosis of early acoustic tumor. He was incapacitated for work, and though I would ordinarily have advised delay until we could be more assured of the diagnosis, he courted the exploration which was made.

Operation revealed a huge posterior arachnoid cystern with thickened and grayish walls. In the right lateral recess a similar condition was found. The nerves in the angle were all brought into full view without disclosing a tumor.

He made a good recovery and remained free from his former attacks for a month. He then had a period of fulness in the suboccipital region with a return of dizziness, which promptly subsided after a lumbar puncture. Since his discharge this experience has been repeated now six times at his home, each time with immediate relief on withdrawal of fluid. He returned here a few days ago to learn what can be done about it. On the whole he is much better as regards his tinnitus and vertigo, and the disk margins are clear. He still has a little nystagmus. Regular saline catharsis is about the best I can suggest for him.

What are we dealing with in this man's case? We can never be certain of course without a postmortem examination, and sometimes not even then, because disturbances of cerebrospinal fluid circulation are particularly difficult to make out when fluid has escaped after the

4. Shall We Decompress for Choked Disk? Tr. Am. Neurol. Assn. **48**:75, 1922.

removal of the calvarium, unless of course there should be such a definite thickening of the arachnoid that it is grossly apparent. "Chronic cysternal arachnoiditis with periodic labyrinthine vertigo" is about as near as we can come to a diagnosis.

Though these are sometimes very baffling cases, experience would tend to show that a large proportion of them may recover if their more critical period is tided over by some palliative operation. It is a subject on which Dr. Horrax will make a detailed report.

These, then, are examples of what come to be listed as "tumor suspects." They represent in many respects our most difficult problems.

UNVERIFIED TUMORS

We come to a group of cases in which there is less uncertainty as regards the underlying cause of the symptoms, tumor being unquestioned. The difficulty now lies in localization and type verification rather than in actual tumor diagnosis. There have been 476 patients with tumor unverified in our past series. Many of them in time will become verified either by operation or necropsy.

There are at present in the wards eleven examples of tumor as yet unverified: five of them have been operated on; the others await their turn. Some patients refuse operation when promise of relief is not great. Many more have deep and inaccessible growths which an operation has failed to disclose. Not infrequently, too, a growth may be exposed which is obviously inoperable and it may seem unwise even to take a fragment for histologic investigation. Some tumors, of course, like most metastatic lesions or the primary pontile tumors, for example, may be regarded, generally speaking, as inoperable, though I shall show an example of the benefit that may be gained by an operation even under each of these most unfavorable circumstances. All these varied factors serve to relegate cases into this unverified group.

At the outset I shall show two or three patients with unquestionable supratentorial tumors who have been benefited by palliative decompressions and who will prove, I think, to any of you who may possibly have come to share Dr. Sachs's views, that a good deal may be accomplished in certain cases even by this somewhat old fashioned measure.

Dr. A. was operated on over six months ago, and though he is not at present a house patient he came in a few days ago to report, to have his eyegrounds examined and his fields taken. He entered the hospital last October on the advice of Dr. J. J. Thomas, because of rapidly advancing choked disk. In the previous July he had had his first severe attack of occipitofrontal headache and dizziness. His discomforts gradually increased until shortly before admission they had become continuous. He had had a good deal of staggering, and during his particularly bad periods experienced some numbness of the left side of the face and tongue. Choked disk had first been observed five weeks before admission. Lumbar puncture was performed: the fluid was negative.

Examination showed merely choked disk measuring about 5 diopters. In addition, he had complete anosmia on the right; slight exophthalmos possibly more marked on the left, and some possible dysmetria of the left upper extremity. Repeated examinations showed nothing more than this.

On October 25, right subtemporal decompression was performed. It was a difficult procedure owing to marked intracranial tension and to unusual vascularity of the bone. An attempt to enter the left temporal ventricle by puncture was unavailing.

He made an excellent recovery from the operation, had no subsequent headaches; the choked disk at the time of discharge November 4, had subsided to 1 diopter, and he left the hospital with a bulging decompression.

He came in a few days ago to report, and shows no change in the former symptoms. The disks are completely flat and though the decompression is full, it is not particularly tense; he states that it is occasionally more so than at present. Right anosmia and slight exophthalmos remain as before. He has been working regularly at his profession, tires easily, but otherwise regards himself as in very good condition.

I may point out that the decompression is about as full as one should ever see after a properly conducted operation. We see a great many people after so-called decompressions who have enormous protrusions due, I think, to misplaced defects and utterly inadequate wound closures. I am dismayed to find that some neurologists are still under the impression that one can properly decompress without opening the dura, and I cannot understand how this idea should have come to prevail. These wounds are closed in four or five layers of delicate interrupted silk sutures for muscle, fascia and galea. The skin sutures have of course nothing whatever to do with the strength of the wound and are merely applied to get the best possible epidermal approximation. Many of the incisions that you will see in the course of the evening, even only ten days after operation, are practically invisible. I may add that invisibility of the scar is something for which every neurologic surgeon should strive, though it is merely an artistic feature of his work.

I may be doing Dr. A. an injustice in letting him go without ventriculograms, but were I in his place I would prefer to go on reasonably content with things as they now stand. I assume that in the course of a year he may begin to have further symptoms, and if his tumor is one that we can actually deal with successfully, this delay will not be in his disfavor.

I shall now show you another case illustrating what I admit is a very unexpected degree of relief afforded by a subtemporal decompression—in what evidently is a pontile tumor.

Case 7 (Walking).—This young fellow is 23 years of age, a linotype operator. Eight months ago he first experienced some difficulty in reading manuscripts, and took to glasses without much benefit. Six months ago he began to have double vision from bilateral oculomotor palsies. One month ago general pressure symptoms set in with severe headaches and vomiting.

Examination showed a high grade of choked disk with hemorrhages. He had also complete paralysis of conjugate movement upward, and though move-

ments of the eyes were possible in other directions, these movements were associated with extreme nystagmus, which is still present. In addition he had marked static instability; was practically bedridden.

It was reasonably certain that there must be a tumor of the pons, and though I did not believe that decompression would help him greatly, it seemed to offer his only chance. In view of his advanced choked disk I did not feel that we were justified in delaying operation even to take a ventriculogram which, if our diagnosis were correct, would merely serve to show dilatation of the ventricles.

A right subtemporal decompression was performed three weeks ago. An exploratory needle was introduced into the temporal horn of the ventricle and air was introduced at the time of the operation. The plates showed, as was expected, a symmetrical and bilateral dilatation of the ventricles of marked degree.

He has shown remarkable and unexpected improvement. His choked disk has rapidly subsided and now measures 2 diopters. He has had no further headache and he walks briskly and steadily. He has actually regained a little upward movement of the eyes, but when he attempts to look up a conjugate strabismus is produced and marked nystagmus elicited. He has had one deep radiotherapeutic treatment. He will be discharged tomorrow with a diagnosis of "Tumor of midbrain: probably glioma: unverified," and will return periodically for observation and further roentgen-ray treatments.

This was good luck, undoubtedly. Generally speaking, subtemporal decompression avails little when there is obstructive hydrocephalus. Moreover, in the presence of a pontile tumor decompression, whether subtemporal or suboccipital, may, by permitting some dislocation of the lesion, actually lead to an increase of preexisting paralyses. The case shows that these rules are not invariable. How enduring will be this young man's period of relief is of course most uncertain.

The next patient shows a still more difficult problem. She, too, has had a decompression long ago and this is a reentry.

CASE 8 (Walking).—Mrs. R. is a healthy-looking woman of 43 who has a typical left hemiplegic gait. She first entered the hospital a year ago, with severe occipitofrontal headache of ten months' duration and frequent attacks of unconsciousness. She then showed full blown choked disks with hemorrhages, poorly-sustained nystagmus, and some unsteadiness of gait. We were unable to agree on a localizing diagnosis. Her vision was threatened, and after one unsuccessful attempt was made to puncture the right ventricle, a right decompression was performed, June 21, by Dr. Horrax as a temporizing measure. She did extremely well. The choked disks subsided with no impairment of vision and she was discharged free from all subjective complaints, with her decompression slightly bulging. There was complete relief for six months.

She reentered the hospital two months ago because of unsteadiness of gait, some return of headaches and the first "unconscious period" (convulsion?) she had had since her discharge.

She showed at this time a peculiar syndrome to which attention will be drawn in connection with a later case. We had a difference of opinion as to whether she had a left cerebellar or a right parietal lobe lesion. She showed on the one hand, nystagmus, a positive Romberg sign, marked unsteadiness of gait, left hypotonus with adiadokokinesis; on the other, a clear-cut left astereognosis with slight sensory changes, and right dorsal toe response.

As events have proved, it would have been best at this juncture to have taken recourse once more to Dr. Dandy's procedures and to have attempted further ventriculographic studies. However, her right parietal region was first explored (April 20), by an osteoplastic operation under local anesthesia with negative findings except for hydrocephalus. The ventricle was easily entered and subsequent pneumograms, if we can interpret them correctly, show what we take to be a filling defect involving both hemispheres, due to a centrally placed growth which gives a mephistophelian appearance to her anteroposterior plates owing to the peculiar flattening and displacement of the ventricles (Figs. 5, 6 and 7).

I may be unduly conservative about ventriculograms. They are oftentimes unnecessary, oftentimes extremely upsetting to the patient,

Fig. 5 (Case 8).—Patient after negative osteoplastic exploration, just before she was shown at the clinic.

often most confusing. Moreover, there has been one unfortunate outcome to the early and overenthusiastic reports: namely, that many neurologists have been led to believe this to be a simple and certain diagnostic procedure which may be undertaken like a lumbar puncture by those possessed of scant surgical training who would not presume to undertake the final operation even should an accurate localizing diagnosis be made. These things, I fear, will lead to a serious reaction against a procedure which, under suitable circumstances as in the case of this patient, might be invaluable.

Experience is gradually teaching us just where we should stand in regard to ventriculography just as it has done in the case of bismuth gastro-intestinal studies. It may serve to localize an increasing number of brain tumors, but for the time being, in the majority of instances at least, it does not replace a thorough neurologic examination.

Fig. 6 (Case 8).—Anteroposterior ventriculogram showing peculiar symmetrical disortion of the ventricles.

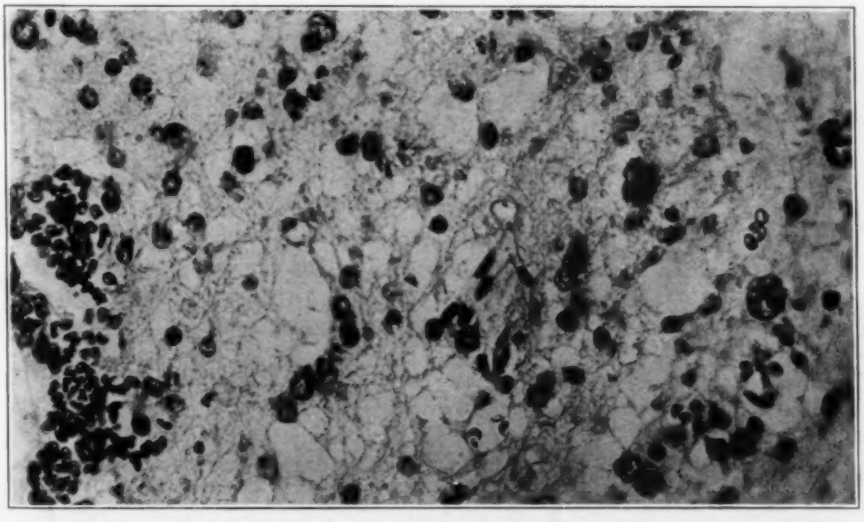

Fig. 7 (Case 8).—Photomicrograph of tumor (cf. Footnote 5). × 300.

Whether we have done right in the case of this woman to tide her along and be satisfied with preservation of vision and avoidance of discomforts, is open to debate. She is cheerful and in good physical condition. Had we known a year ago just where her lesion lay and attacked it, I do not believe she would have been any better off. If it is a benign lesion like an endothelioma we may still be able to remove it. If it is a glioma we could not have removed it in its totality a year ago, even had we been able then to localize it.[5]

There are two other patients at present in the hospital with problems almost as difficult as that laid bare in the foregoing recital. The first of them is blind and disoriented. I shall give the main incidents of his history without bringing him here.

Case 9.—This man, 48 years of age, during the past year has made the rounds of many clinics and his history is a complicated and difficult one to untangle. He has had tinnitus for twenty-five years and has become almost deaf in the right ear. Naturally it has been suggested that he might have an acoustic tumor. He has had extraordinary periods of prolonged comatose sleep associated with prostrating headaches, hiccoughing and vomiting. From some of these attacks he has had sudden waves of improvement. A year ago he was in the Mayo Clinic in such bad condition that an operation was thought inadvisable. He left for home on a stretcher; suddenly improved, and on reaching home was well enough to walk off the train unaided. He remained

5. *Subsequent Note.*—On June 8, another attempt was made to disclose this woman's tumor. It could be detected by a blunt exploratory needle deep in the hemisphere. The appearance of the ventriculograms, showing as they did a filling defect of the ventricle on each side, made it seem probable that the growth was median; and consequently bone was removed up to the longitudinal sinus so that it was possible to look between the hemisphere and the falx, and at a depth of about 4 cm. the upper surface of what was taken to be an endothelioma was disclosed lying astride the margin of the falx. The patient's condition did not justify an attempt at its removal. It was impossible to replace the boneflap, so that it had to be stripped away to permit a closure. She did remarkably well after this step—so well, in fact, that I determined to make a transcortical incision in an attempt to remove the growth. This was done June 13. It proved to be a huge frontal glioma which was growing into the ventricle. A mass of the tumor, as large as a fist and weighing 150 gm., was removed. She again made an excellent surgical recovery and at the time of discharge, July 21, was up and about on her feet and rapidly regaining the use of her arm; free from discomforts, with normal vision and with her decompression area flat. The tumor is what we designate a protoplasmic glioma (Fig. 7).

From the first we were utterly at sea in this woman's case misled by the clinical symptoms as well as by the ventriculograms. The decompression, with its nine months' period of complete relief, is possibly the best thing that could have been done for her at that time and unquestionably saved her vision. She now promises, after all our struggles, to do extremely well and will have further roentgen-ray treatments, for we think a tumor of this type is particularly amenable to deep radiation.

practically free from symptoms until the following September. He was brought here a month ago in one of his stuporous periods.

Examination showed receding choked disks, with near blindness; diminished audition on the right; positive Romberg sign.

One would feel that in a case of this sort a ventriculogram was likely to give more information than anything else, and consequently the right hemisphere was tapped with the expectation of finding a dilated ventricle. As a matter of fact the ventricle contained only a small amount of fluid, which was xanthochromic; and an attempt to introduce air for the purpose of getting a ventriculogram was futile. Consequently an immediate subtemporal decompression was performed.

He has improved greatly, but is still bedridden, blind, and nearly deaf.

Fig. 8 (Case 9).—Glioma of third ventricle; natural size.

I have not the slightest idea where the tumor is, but it certainly is above the tentorium and, in view of the xanthochromia, probably involves the ventricles.[6]

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6. *Subsequent Note.*—This man's condition remained unchanged for weeks. On July 5, in despair, under local anesthesia the left ventricle was punctured and found to be dilated with xanthochromic fluid. A ventriculogram showed that the air did not pass from the left to the right ventricle. Following the puncture the pressure symptoms, bad enough before, became greatly aggravated and though the air was promptly removed he died suddenly about twelve hours later. The necropsy showed a huge glioma of the third ventricle (Fig. 8).

There is still another among the unverified tumors in the house that presents a problem of equal interest, though there is less difficulty in localization. She also is too uncomfortable to bring here.

Case 10.—This patient is a young woman of 20, a stenographer, who came on March 21, with a diagnosis of cerebellar tumor. Her illness dates from an attack of influenza fourteen months ago. She subsequently lost a good deal of weight, and in August, 1922, began having severe and almost constant headaches with almost daily projectile vomiting. Three weeks before admission she developed left-sided internal squint; at that time also choked disk of low grade was first observed.

On examination she showed marked cerebellar symptoms with sustained nystagmus on looking to right and left as well as upward; positive Romberg sign with falling to the left and backward; definite deviation to the left in

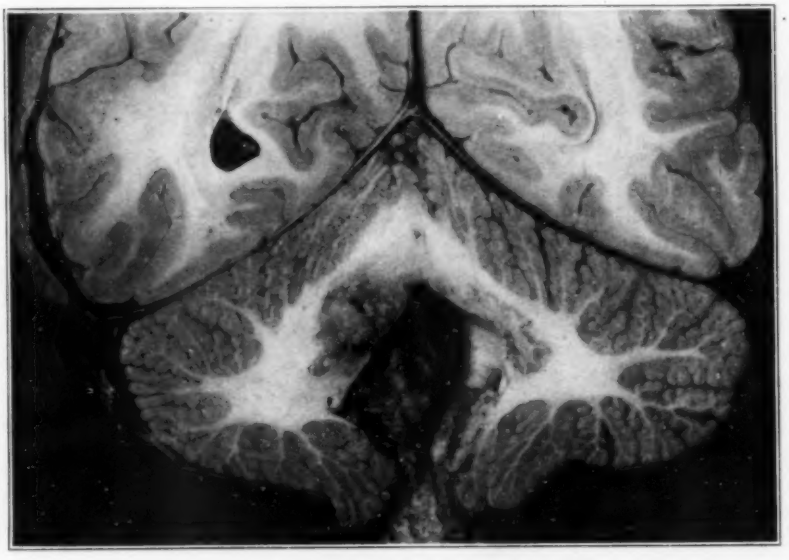

Fig. 9 (Case 10).—Tuberculoma projecting into dilated fourth ventricle; natural size.

walking; partial left facial palsy; weakness of the left abducens and deviation of the protruded tongue to the left. It may be added that Dr. H. H. James who sent her here found that she had a positive intradermal tuberculin reaction.

These were the essential findings. In spite of the absence of any impairment of hearing it was thought that she probably had an intracerebellar tumor on the left side extending into the recess.

She was kept under observation for three weeks before operation. It was undertaken only because she had begun to show advancing choked disk. The cerebellar exploration was negative. There was no special tension of the dura, the cranial nerves on the left side were all seen, and though there was a somewhat peculiar projection of a nodule of cerebellum between them, it was not identified as tumor and the wound was closed with a possible diagnosis of pontile tumor, or chronic arachnoiditis.

She recovered well from the operation, but it had no beneficial effect on her symptoms. She has gradually gone down hill during the succeeding three weeks, with persistent vomiting, almost perpetual nystagmus and of late such extreme vertigo that she is unable to turn from her left side. In addition, she has lately begun to show a good deal of incoordination of the left arm and leg and the choked disk has advanced though the edema has never been high.

This woman, therefore, has an unverified lesion, presumably of the brain stem. A tuberculoma perhaps, but more probably a glioma. The lesion has apparently caused a nuclear palsy of the seventh and sixth nerves on the left, and has involved the vestibular nucleus as well. The eighth and fifth nerves have escaped. The wound is perfectly healed, and if we should discharge her it would be better for our surgical statistics, for it is a regulation that every case dying in the hospital after an operation is recorded as a postoperative fatality no matter how long the interval may be.[7] We are, however, more interested in learning something from our mistakes than in improving our operative percentages, and I am glad to say that even on this basis our fatalities do not range much over 15 per cent.

CASE 11 (Bed case).—This poor man is 43. His symptoms began with loss of vision, lack of ambition, and frontal headaches, eighteen months ago. He is now quite somnolent. His vision at present is reduced to shadows in the left eye and 10/100 in the right. The disks are pale and slightly edematous, and there has been a difference of opinion as to whether the edema is superimposed on a primary atrophy or whether it is a secondary atrophy. He has marked static instability, incoordination of right arm and leg with definite hypotonia; no nystagmus.

It is the sort of case in which there is great difficulty in distinguishing between a median cerebellar and a suprasellar lesion. He has had ventriculograms made and they merely show symmetrical dilatation of both ventricles.

One thing is distinctly in favor of a supratentorial lesion: namely, some fields of vision which were taken by a competent ophthalmologist a year ago and which then showed a lower right quadrantal homonymous defect. This we would ordinarily have regarded as evidence of a left temporal lobe lesion but in view of the complete dilatation of the temporal horn as shown by the ventriculogram, this past finding must, I presume, be disregarded.

In addition, he shows a subnormal temperature, often as low as 97, and a basal metabolism of —20 per cent. Though an internal hydrocephalus may give similar reactions, this sounds more like a pituitary or suprasellar lesion. However, the sella is normal and there are no suprasellar shadows.

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7. *Subsequent Note.*—She began having fever on May 30, passed into coma and delirium, and died June 3. Examination of the brain revealed marked hydrocephalus produced by a local lesion of unusual site and appearance (Fig. 9). A nodular growth the size of a hickory nut projected from the brachium pontis on the left side of the dilated fourth ventricle. It involved the restiform body of the cerebellum, and must have caught principally the left vestibular nucleus as well as the nucleus of the left abducens and the loop of facial fibers which embrace it. Diagnosis: "Tuberculoma of brain stem: verified."

I presume that this problem in differential diagnosis is one which oftentimes confronts many of you. To our untutored eyes it is sometimes impossible to distinguish between primary atrophy with superimposed edema and choked disk subsiding with atrophy. His symmetrical hydrocephalus might be produced either by a lesion above or below the tentorium, and though nystagmus is absent he has some fairly characteristic cerebellar symptoms. Just what I shall do I do not quite know, and among my junior colleagues opinion is divided. I rather incline toward a left transfrontal exploration which would expose the chiasm as well as the tip of the temporal lobe.[8]

We now come to a series of four other cases not yet operated on—all of them however with symptoms which permit of a fairly definite

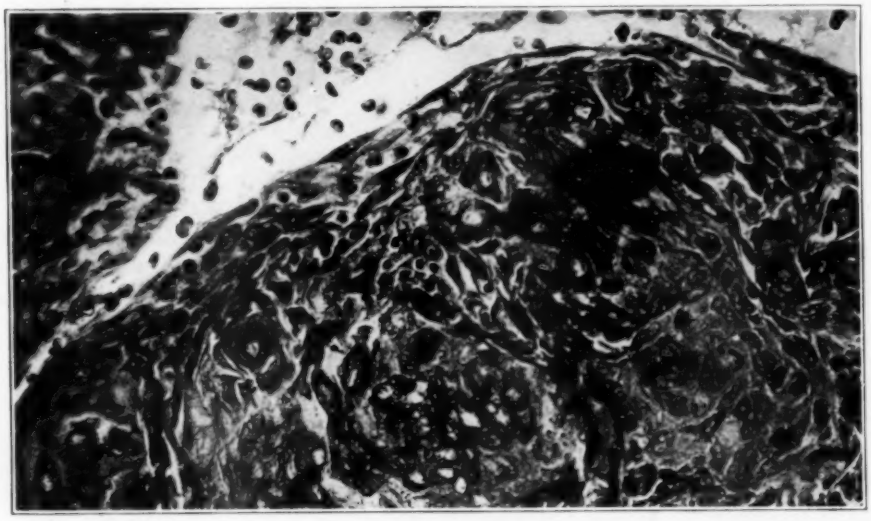

Fig. 10 (Case 11).—Portion of wall of pharyngeal pouch cyst. × 300.

localizing diagnosis. All four are children, and I shall begin with the youngest.

8. *Subsequent Note.*—We agonized over this man until June 16 when a left transfrontal operation was performed. The chiasm and nerves appeared to be normal though there was so much thickening of the arachnoid I thought for a time a chronic arachnoiditis might be the cause. The temporal lobe appeared normal. With the intent of puncturing the third ventricle to relieve the hydrocephalus, the posterior margin of the chiasm was exposed and a brain needle introduced. Instead of clear fluid it struck coffee-colored fluid rich in cholesterin crystals—evidently a pharyngeal pouch cyst. A portion of its wall was removed for verification (Fig. 10). (I have never before encountered one of these cysts in this posterior situation.) From a dull somnolent state he was transformed to an alert individual, and made an excellent recovery, in spite of a wave of postoperative polyuria accompanied by nausea and inappetence.

CASE 12 (Bed case).—This boy, six years of age, previously well, was sent home from school only a month ago (April 16) because of a bad frontal headache. Headaches with vomiting persisted for the next two weeks, and soon some weakness of his left arm, leg and face was observed. He also complained of pain in the region of the left hip. He became progressively worse and there has been rapid failure of vision. He was brought here in an ambulance a week ago. His symptoms, therefore, have been of only a month's duration though it is possible that he may have been having some difficulty with vision for a longer period.

The most striking neurologic findings are: (1) Extensive oculomotor palsy, there being practically no lateral conjugate movements except on the part of

Fig. 11.—Tumor in a patient with symptoms comparable to those of Case 12. Note the herniation from the affected hemisphere compromising the pons.

the left internal rectus. Vertical movements are normal and without nystagmus. (2) Choked disk of from 3 to 6 diopters, receding with atrophy and loss of vision. (3) Left hemihypesthesia and hemiparesis, the arm being completely flail. Indeed, the muscles throughout are so markedly hypotonic that deep reflexes are difficult to elicit, but they are probably increased on the left. (4) His obviously enlarged head, which has cracked-pot resonance on percussion, due to the separated sutures that show in the roentgenogram. It is a condition common in hydrocephalus and suggests a posterior lesion. He has, moreover, extreme suboccipital tenderness to pressure.

There have been differences of opinion about this little boy. One would feel that his oculomotor disturbances point with certainty to a pontile lesion with nuclear palsies. (This opinion was concurred in by members of the Association). If this is so, he probably has a hydrocephalus, but there is a possibility which, in spite of the extreme hypotonus, his hemiparesis suggests; namely, that the lesion may be a large

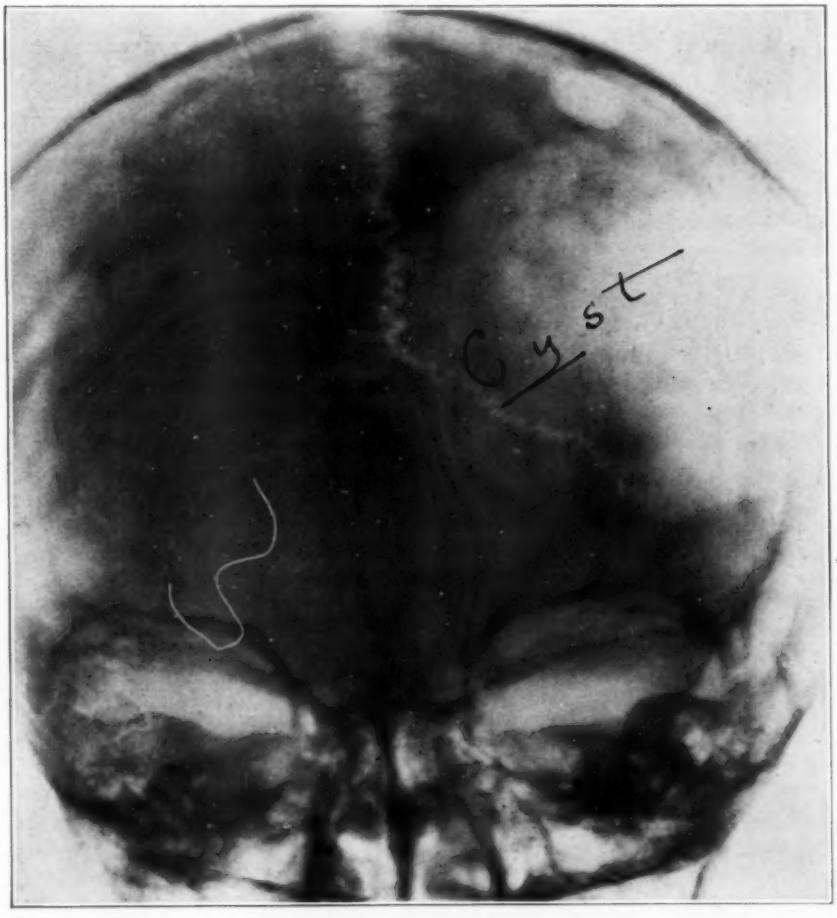

Fig. 12 (Case 12).—Cranial pneumogram showing extent of huge gliomatous cyst.

one in the right hemisphere. The fields of vision might determine this, but he is too young, too incooperative and too nearly blind to secure dependable charts. I will pass about a photograph (Fig. 11) which shows the brain of a child, seen many years ago, who had a very similar syndrome, the supposedly pontile and false localizing symptoms having been due to pressure on the anterior pons by a cerebral hernia-

tion through the incisura tentorii, provoked by the huge tumor of the hemisphere. This child, I may add, was supposed to have a cerebellar lesion and it is just the sort of case in which ventriculography would be of great value.[9]

Cases notoriously run in groups, and there are three young girls, supposedly with cerebellar tumor, who have been admitted during the past week. All of them urgently need operation if we are to preserve vision.

CASE 13 (Walking).—This child, Hazel McD., 12 years of age, was admitted May 25, 1923. She looks the picture of health but for the past year has been having spells of dizziness and vomiting in the early morning which have increased in frequency. Accompanying the more severe of these attacks there has been pain referred to the neck and shoulders. Her symptoms, as so often

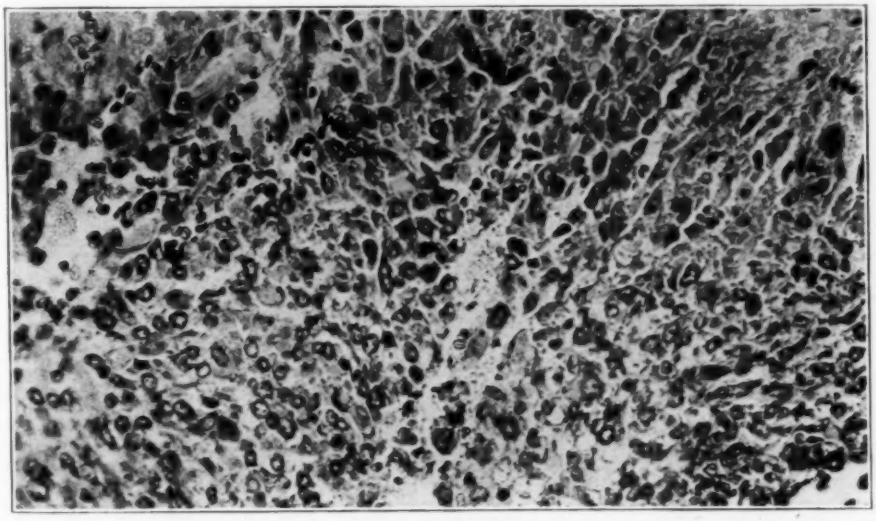

Fig. 13 (Case 12).—The histologic character of the tumor. × 300.

9. —*Subsequent Note.*—To determine whether there was tumor or a dilated ventricle in the right brain, a puncture was made on June 4. The needle entered a huge gliomatous cyst from which 100 c.c. of yellow clotting fluid was withdrawn and replaced by 80 c.c. of air. The roentgenogram (Fig. 12) gives some idea of the size of the cyst. On June 7 an osteoplastic resection was made; the cyst was opened, its walls were fixed with formalin, and the only nodule of tumor which could be detected was removed from the cyst wall. It showed a glioma. The child almost immediately began to regain the use of his paralyzed side after the puncture and did well for a few weeks but symptoms again returned and the flap became raised. Consequently on July 16 the flap was re-elevated. The position of the cyst was occupied by a huge tumor mass of whose presence there had been no indication before. It was removed *en bloc*. The mass removed weighed 144 gm. Histologically, it shows (Fig. 13) a non-fibrillar glioma of peculiar character. The boy (except for his unfortunate loss of vision) had almost completely recovered at the time of his discharge August 12.

happens, were at first thought to be due to some gastric trouble and gastrointestinal studies were interpreted as showing stasis of the ileocecal junction. Consequently, a few weeks before her admission an exploratory laparotomy was performed.

From the outset her vomiting has been projectile. For a month she has been having blurring of vision, diplopia, and increased suboccipital discomforts.

There is a choked disk of 5 diopters. She holds her head to the right in a somewhat fixed attitude. There is considerable hypotonia of the whole muscular system, but without special incoordination of movement. With eyes closed there is definite swaying, but the usual tests to elicit cerebellar incoordination are fairly well performed. There is possibly some clumsiness of the left hand and arm. We find no other symptoms worthy of note.

It is our impression that she has a cerebellar lesion, though there is very little to show for it and I do not wonder that her former attendant

Fig. 14 (Case 14).—Photograph (after operation) showing the fixed position of the head.

was led to explore the abdomen—far as it is from her cerebellum. She may possibly have an ependymal glioma of the fourth ventricle, for Dr. Bailey's analysis has shown that vomiting was an early symptom in all of the cases.[10]

Case 14 (Bed case).—Elizabeth McC., a schoolgirl of 13, was perfectly well until February 12, when she fell and struck her head behind the right ear.

10. *Subsequent Note.*—Operation was not performed until June 18. Aside from tension and marked herniation of the cerebellum into the spinal canal and internal hydrocephalus, no lesion was disclosed. She did extremely well and was discharged July 7, her choked disks completely subsided. She was given one treatment with deep radiation and will return periodically for others. Diagnosis: "Intra cerebellar tumor (glioma?), unverified."

Two weeks later she began having attacks of vomiting which were thought to be bilious and she had some transient oculomotor symptoms. These symptoms subsided; she was sent back to school and was fairly well, though occasionally dizzy and nauseated, until three weeks ago. At that time double vision was again observed and with it considerable loss in visual acuity. Suboccipital discomforts appeared, with nausea, vomiting, and staggering.

About the same time she began to hold her head to the right and she has had a number of "sinking spells" in which the eyes roll upward, and the arms become flexed and rigid. These spells are inaugurated by a feeling of numbness of the face and teeth and followed by a long period of somnolence. Lumbar puncture was performed shortly before her entrance, without upsetting her.

Examination shows an early choked disk of 2 diopters, coarse nystagmus both horizontal and vertical; left abducens paralysis—indeed a paralysis of conjugate movement to the left in both eyes; crossed hypesthesia involving the right side of the body and left trigeminal area where there is corneal areflexia. There is also a marked Romberg sign, staggering gait with tendency to fall to the right, and some incoordination of the extremities. Perhaps the most striking symptom is the fixed position in which she holds the head (cf. Fig. 14).

Roentgenograms show nothing more than evidence of intracranial tension. The deep reflexes are brisk, definitely more so on the right where there is a positive Babinski sign.

There can be little doubt of the approximate position of the lesion, and I think all will agree in the diagnosis of a pontile tumor with secondary hydracephalus. It is in cases of this sort that one may see outspoken paralyses without a very high grade of choked disk: in short, the localizing symptoms as a rule far outweigh the general pressure symptoms.[11]

CASE 15 (Walking).—E. H. G., aged 16, had had symptoms since December, 1922, beginning with headaches, slight fever, tinnitus, and convergent squint. Naturally she was thought to have encephalitis. During February, March, and April she was apparently well and was again at school: but for the past month severe frontal headaches have reappeared, some dimness of vision has been noted and there is diplopia on looking to the right.

She is an intelligent and normal appearing girl. It is hard to believe that she has a high grade of choked disk with hemorrhages, and that the roentgenograms showed increased intracranial pressure. She sways a little with her eyes closed, uses her left hand a little clumsily; there are a few nystagmoid twitches to the right and weakness of the right abducens. In brief, here is a

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11. *Subsequent Note.*—June 5, a suboccipital exploration was made by Dr. Horrax. There was internal hydrocephalus, moderate pressure cone, a somewhat enlarged cerebellar hemisphere; otherwise a negative exploration. The child did extremely well as a result of this decompression and was soon able to sit up. The choked disks rapidly subsided, and the discomforts were relieved —no further vomiting, no headaches. At the time of her discharge, June 21, the local paralyses remained much as before. She was given deep radiotherapy. The case remains as yet "pontile tumor: unverified: tuberculoma or glioma?"

child with little more than headaches and advanced choked disk, and we have to stretch matters to read any localizing symptoms into her case.[12]

I do not know what we shall find in any of these three children. All of them, I assume, will have suboccipital explorations—the sooner the better so far as vision is concerned—and if the findings are negative supplementary roentgen-ray treatments will be given.

In the same ward with them is a girl who was here a year ago in a condition similar to, perhaps even worse than, theirs; at her operation a deep gliomatous cyst was punctured. She will serve as a transition from the unverified to the verified tumor cases to follow.

Case 16 (Walking).—Sarah H., is 12 years old—a healthy looking girl. She entered the hospital two years ago with a history not unlike that of the children just presented—eight months of headaches, vomiting, stiffness of the neck, unsteadiness in gait, and, alas! marked lowering of vision. She showed the usual typical cerebellar syndrome—suboccipital tenderness, hydrocephalus, choked disks, nystagmus and motor incoordination with static instability, adiakokinesis, asynergia, hypermetria, hypotonus and all the rest.

At operation nothing was found on the cerebellar surface. A puncture of each hemisphere entered a small gliomatous cyst—perhaps two cysts in the same tumor—containing about 10 c.c. of yellow clotting fluid. She made an excellent recovery, except that we were not in time to save her vision. She could only distinguish shadows on the subsidence of her choked disks. She was given deep roentgen-ray treatments.

She has just returned for an examination in response to a letter of inquiry after two years. She is a little unsteady but gets about very well. Her neck is a trifle full, though only a surgeon would realize it. The disks are flat. She looks in perfect health, but will be given another deep cerebellar radiation while here.

VERIFIED TUMORS

We now come to the verified cases. I may call attention again to Table 1 and to the main subdivisions under which the cases are listed. Of most of these groups, in sequence, I shall show some examples, reserving the gliomas to the end as they represent the largest group and surgically the most difficult cases with which we have to deal.

THE ADENOMAS

Needless to say, these are largely hypophysial in origin. The disease, which was unknown twenty years ago, we now recognize about as easily

12. *Subsequent Note.*—During the next ten days the nystagmus became slightly more pronounced: right corneal areflexia with slight hypesthesia of the right face was detected: also some slight incoordination of the right hand on the usual tests. There was right suboccipital tenderness, and possibly some loss of acoustic acuity on the right. A possible recess tumor was suspected. The suboccipital exploration on June 12 was entirely negative except for hydrocephalus. She has done well as a result of the decompression; the headaches were relieved and on her discharge, July 19, after two deep radiotherapeutic treatments, the choked disks were flat with vision preserved. Diagnosis: "Cerebellar tumor (Glioma?): unverified."

as thyroid disease, and it is probably as common. Sadly enough we know little about the underlying factors which provoke these pituitary tumefactions. We do well enough perhaps to recognize them at all and are called on to operate with the chief object of preserving vision. In this regard we stand today in relation to these hypophysial "goiters" in much the same position that the surgeons stood who first began to operate for thyroid goiters because of deformation of the trachea and consequent dyspnea.

Hypophysial adenomas are often attended by cephalalgia at an early stage, but it is not uncommon for the discomforts to cease before extrasellar pressure disturbances begin to appear. In other words, subjective relief is apt to be experienced by the time the tumor has fully distended the sella and burst its fibrous capsule. The growth may extend through the dura into the intracranial chamber or downward into the sphenoidal cells; or, indeed, in both directions.

There are two possibilities of approach. I shall give examples of each of them. There are only two verified adenomas at present in the house. Both happen to be ones which have been operated on by the transfrontal rather than transsphenoidal route—that is, by an exposure from above through an osteoplastic operation rather than by one through the nose. In neither of them has the result been as satisfactory as I could wish in so far as rapid improvement in vision is concerned. In both cases the process was long standing, and we perhaps should not expect much more than to hold the vision where it now stands.

CASE 17 (Walking).—Mr. S., 48 years of age, is a bookkeeper who shows a bad scar on the left side of his forehead. This is not his operative scar, which is on the right side. The other scar was made by a horse's hoof when he was a child. Whether it has any bearing on his trouble I do not know.

Mr. S., enjoyed good health till about eighteen months ago, when vision began to fail and repeated change of glasses gave no relief. He complains too of excessive drowsiness and of growing stout. He had never had headaches. This is about all. I do not think that one would pick him out on the street as an example of adult hypopituitarism though ordinarily the condition is about as easy of recognition as thyroid myxedema, and I may say it is a far more common malady. There was little noteworthy in his examination, except the primary optic atrophy, an unusually clear-cut bitemporal hemianopia with vertical meridians, and slight anosmia on one side. The basal metabolism was normal. The roentgenogram showed a widely distended sella with unusually well marked diploetic channels in the cranial bones. Owing to this and the very peculiar absorption of the anterior clinoid processes, I inclined to a diagnosis of suprasellar meningioma. Dr. Bailey thought he had a congenital lesion, and Dr. Sosman, the roentgenologist, a pituitary adenoma. Dr. Sosman's view was correct, and had I accepted it, Mr. S. would have had a transsphenoidal operation and been home two weeks ago. I thought it was best, in view of the difference of opinion, to get a look at the lesion from above. It proved to be a very large adenoma which had crowded its way upward between the legs

of the chiasm and had largely absorbed the anterior clinoids which were paper-thin and tilted upward. It had not burst through the capsule and, though under these circumstances I dislike to operate from above, the capsule was incised and large masses of the relatively nonvascular growth were scooped out of the fossa until the stretched nerves and chiasm lay collapsed and free. It proved to be a typical pituitary chromophobe adenoma.

His fields have showed no change, though I hope they may still improve. He has had two deep therapeutic radiations. His metabolism two days ago was —11 per cent. The scar, as you see, is practically invisible. He is taking pituitary extract (whole gland) and will be discharged in a day or two, to report in a few months.

The next patient has a history extending back many years, which began with separation from her husband twenty-one years ago; increasing blindness for eighteen years; convulsions for fourteen years. She has had prolonged antisyphilitic treatments.

Case 18.—Mrs. I., is a woman, aged 60, who has the look of pituitary insufficiency. She began to lose her vision twenty years ago and the left eye soon became completely blind. The other eye was also much impaired but vision remained nearly stationary until twelve months ago, since when this eye also has been failing. For many years she suffered greatly from headaches, but these of late have been less annoying. For a long time she had also had curious fainting attacks, associated with polyuria, incontinence and loss of consciousness. She has been taking a patent medicine for some years to control them.

Examination showed primary optic atrophy; a clean-cut temporal hemianopia in her right (the seeing eye) where vision is reduced to 20/200; a moderately distended sella with eroded clinoids; metabolism of —10 per cent., with the usual accompaniment of subnormal temperature, slow pulse, moderate adiposity, and so on. She shows the usual pallid skin and loss of hirsutes characterizing the malady.

On admission drugs were stopped and shortly she had one of her familiar attacks, fortunately without loss of consciousness or convulsion. It began with the peculiar numb feeling which always accompanies them, and during the course of an hour she passed 1,800 c.c. of urine and had two watery bowel movements.

This peculiar circumstance led me to feel that she in all probability had an adenoma which had penetrated the dural capsule and was involving the infundibular region. Otherwise I would have preferred to operate from below.

A transfrontal operation, the scar of which is barely visible, was performed May 12. The optic nerves were found widely separated by a soft bulging adenoma which, however, had not pushed through the dural capsule. This was incised and a considerable portion of the soft tumor was spooned away. It proved to be a typical chromophobe adenoma.

She has made a good operative recovery and her visual acuity has improved to 20/100, but there has been as yet no widening of visual fields, and we possibly cannot expect any. She may have to be satisfied after eighteen years of pressure on the nerves if she can retain the vision she now has.

Lest you carry away the impression that these two last patients typify the outcome of the usual operation for a pituitary adenoma, I

shall show a more encouraging example. It is a two-year result after a transsphenoidal operation through the nose. The man came in a few days ago, as he does every six months, to have his fields taken.

Mr. C., 50 years of age, has long been a clerk of our Supreme Court—a position for which good vision is as essential in Massachusetts as elsewhere. He is a vigorous looking man, in spite of the fact that in the last twenty years he has advanced from 150 to 230 pounds (from 68 to 94 kg.). On the advice of Dr. J. J. Thomas he entered the hospital two years ago, with the story that for five years he had gradually been losing vision until for three months he had no longer been able to read the news sheets. Coincidentally there had been frontal headaches and for a year rapid increase in weight.

The examination showed primary optic atrophy, bitemporal hemianopia (Fig. 15, *A*), adiposity, a large sella, and a basal metabolism of —10 per cent.

It was regarded as a suitable case for a transsphenoidal operation. This was performed on April 5, 1921. The tumor was found filling the sphenoidal cells where, having burst through the sellar floor and dural capsule, it was covered only by mucous membrane. A large amount of the soft struma was cleared away.

He experienced subjective improvement in vision on recovering from the anesthetic; and four days later the perimeter recorded vast improvement in his fields (Fig. 15 *B*).

He was discharged April 16, eleven days after the operation, with visual fields normal (Fig. 15 *C*). The acuity in the eye most seriously affected showed improvement from 20/200 to 20/50.

Since then, to use his own words, he has not lost a day's work; he is free from headaches, with normal vision and, as formerly, he usually reads every night till after midnight.

The vision, uncorrected, is now 20/30 in each eye, but the fields, which have remained practically normal for two years (Fig. 16 *A* and *B*), now show for the first time a little notching of the upper temporal quadrants (Fig. 16, *C*). In view of this he will be given a deep radiation.

This was a fortunate case, I admit, but our pituitary series contains many others equally satisfactory. No transfrontal operation at the best can quite do this for a chromophobe adenoma. In most cases, blood stilling from the raw surface of the gland is the serious problem; and in operating from above one runs some risk, in spite of all care, of injuring the nerves. Then, too, one can never be sure whether the adenoma, as in Mr. C.'s case, has already burst its dural and bony capsule and extended into the sphenoidal cells. Of the 169 cases listed in the table, 164 have been operated on, 149 by the transsphenoidal route, nineteen by the transfrontal route, and four by both routes. There have been fourteen postoperative fatalities after the former, and one after the latter procedure. The mortality, therefore, is higher for the transsphenoidal operation (9.3 per cent.) than for the transfrontal (5.2 per cent.), but this is partly due to the fact that the earlier operations for advanced cases were done in this way, and two of the fatalities were the result of inadvisable second or third operations when the first had failed to relieve

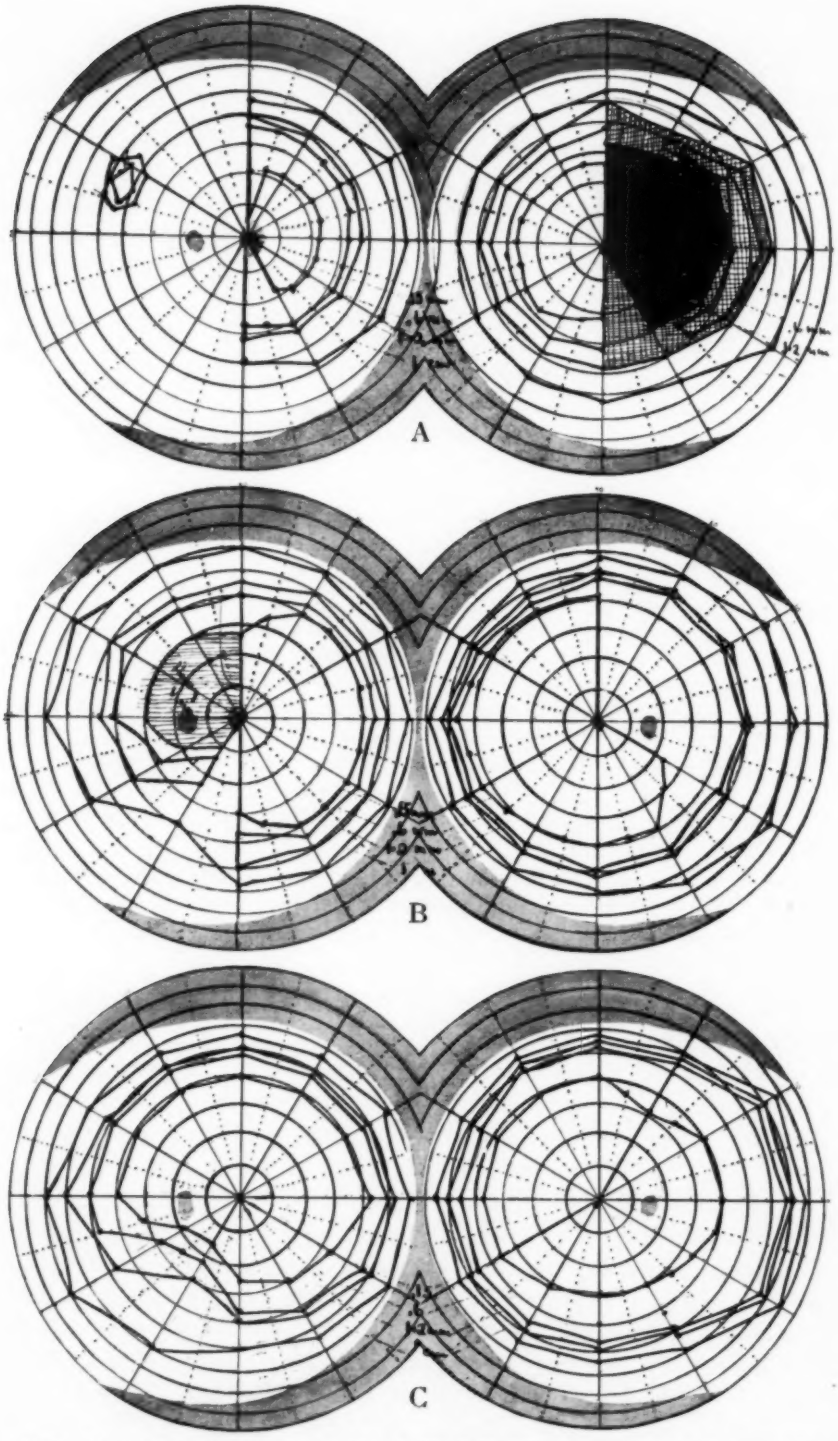

Fig. 15.—Fields of vision. *A:* March 30, 1921. Preoperative. V.O.S., 20/200; V.O.D., 20/30. *B:* April 9, 1921, four days after operation. V.O.S., 20/50; V.O.D., 20/20. *C:* April 16, 1921, eleven days after operation. V.O.S., 20/50; V.O.D., 20/20.

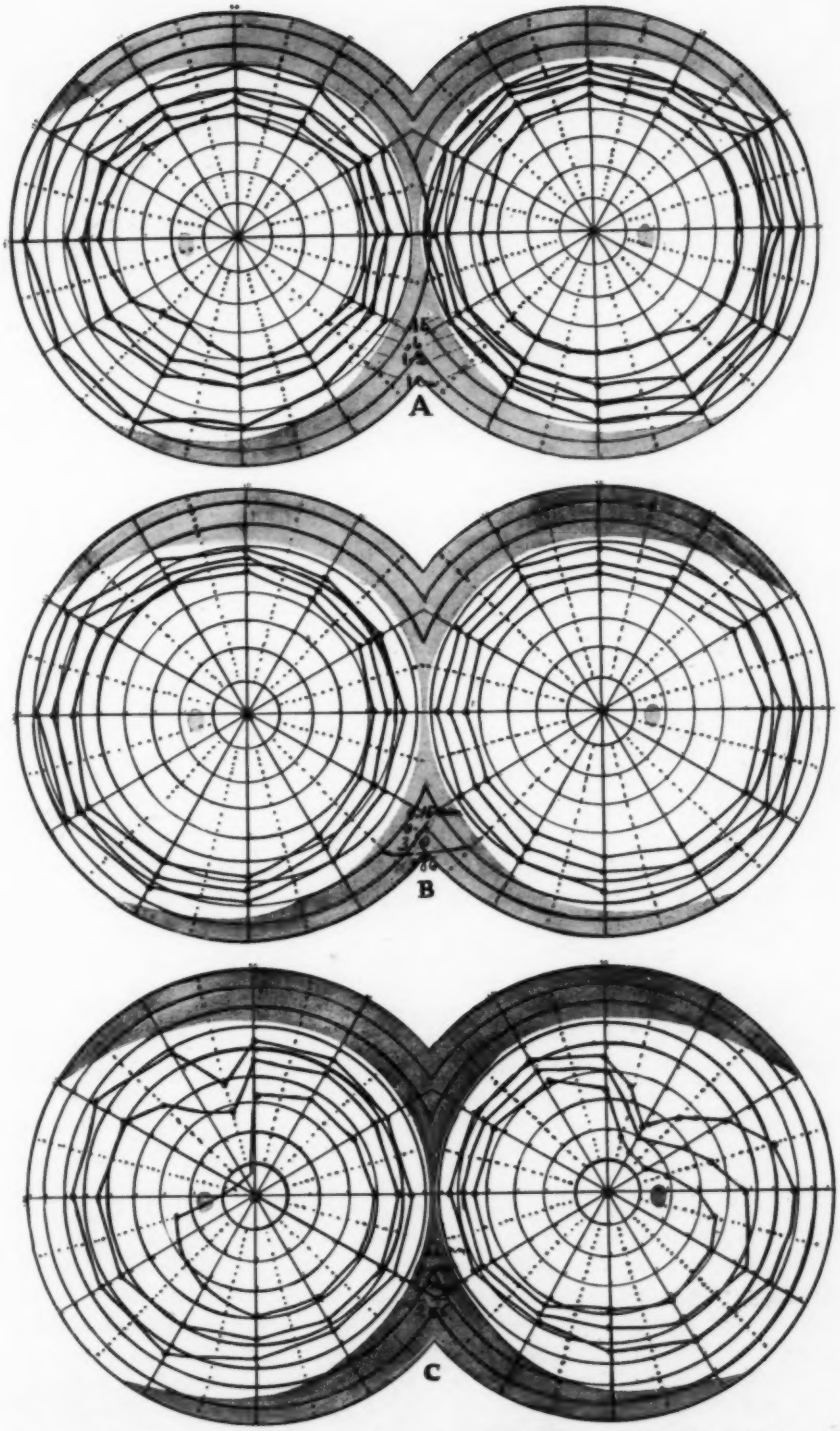

Fig.16.—Field of vision. *A:* May 9, 1921; four weeks after discharge. V.O.S., 20/40 +; V.O.D., 20/20 —. *B:* Nov. 15, 1922—after eighteen months. V.O.S. corrected, 20/30; V.O.D. corrected, 20/20 —. *C:* May 23, 1923. V.O.S., 20/30; V.O.D., 20/30.

vision. The case mortality has been 9.1 per cent. for the entire series; the operative mortality 8.2 per cent. The end results are the important things, but this would lead me too far astray.

THE MENINGIOMAS

These have been made the subject of a recent Cavendish lecture in which an apologia was made for assigning to the tumor commonly called dural endothelioma a new name. Their source of origin from the arachnoid, their association with the arachnoid villi and consequently with the dural sinuses, was then emphasized.

Though unmistakable lesions in the long run, they are of variable histology ranging all the way from very cellular, rapidly-growing, sarcoma-like growths, to fibrous lesions full of psammona bodies. They need to be carefully studied from a histogenetic standpoint, for it is still undetermined whether mesothelial elements are capable of transformation into fibroblasts.

There are two verified examples at present in the house. The first of them illustrates the tendency of the more rapidly growing types of these tumors to recur if broken or incompletely removed at the first session.

CASE 19 (Walking).—Mr. T., a school teacher, was 39 years of age when he first entered the hospital in February, 1916. For eight months he had been having increasing disability of the left leg, first noticed as a lack of control over movements of the toes. Soon there began to be some numbness of the left hand and arm. Subsequently there were many jacksonian attacks beginning in the arm which for a month before his entrance had become practically useless.

He had had no headaches though he was aware of a sense of intracranial fulness. He had never lost consciousness in one of his attacks; they were invariably preceded by numbness of the arm, and indeed the focal convulsions, of which he had had only three, were limited to the arm and to the left corner of the mouth. The leg and foot had never been involved. It would have been a little more usual for the focal attacks to have preceded the paralysis which began in his leg, rather than to have followed it.

Examination showed left-sided hemiparesis, with increased deep reflexes and dorsal toe response.

Although there were no pressure symptoms whatsoever, the story was sufficiently typical of tumor. In view of this, it is interesting to read in his hospital history an account of previous treatments by six successive physicians before he came into the hands of some one who suggested tumor.

I find that my own recorded diagnosis seven years ago favored a right parasagittal glioma, but at the operation an endothelioma was found and removed at a single session. The tumor was a fairly large one, weighing 98 grams. In its extirpation I did a very unwise thing. For, when the growth had been exposed and partly enucleated I found that it partially underlay the upper margin of the osteoplastic defect. Consequently in dislodging the growth, it was fractured and a small fragment of it might easily enough have been left

behind. To ensure complete removal, I should have rongeured away the shelf of bone up to and beyond the sinus. But this would have necessitated a two-stage operation with sacrifice of the boneflap, for reasons the surgeons will understand, and I was glad enough to get out of my difficulties even though there was a possibility of incomplete extirpation.

Mr. T., made an excellent recovery, completely regained the use of arm and leg, and with the exception of an occasional mild focal attack remained perfectly well until a few months ago—a period of seven years. This past winter he began to notice a return of weakness in the left leg. There was also some headache and he had numerous jacksonian attacks involving arm and face but with no loss of consciousness. He fought against these things and kept at his studies till the left side had become so weak and spastic he could barely get about. Hearing of his plight and knowing what was going on in all certainty, I urged him to return.

Under local anesthesia, in two stages, on May 1 and May 4, this recurrent tumor was removed. It necessitated leaving a considerable defect in the midline over the longitudinal sinus where I found the bone involved, though the roentgen rays gave no indication of it. I had the unusual experience of finding the sagittal sinus occluded by tumor and an unattached plug of growth 3 cm. in length was removed from within the sinus which subsequently was resutured. With the addition of this fragment I think the growth is completely intact.*

He is rapidly recovering; indeed, the afternoon of the operation he was able to move his toes—for the first time in eight months. Immediate improvement in function rather than further loss may be taken in intracranial as well as intraspinal surgery as an evidence of the delicacy with which a tumor is removed and the degree the nervous tissues are spared from trauma in the process.

The chief lesson to be drawn from this case is obvious: namely, that one cannot be sure there will be no recurrence after the removal of a meningioma unless the growth is removed intact and with it the entire dural area to which it has become attached.

These mesothelial growths behave in most peculiar ways, for not only may the dura become invaded but the adjacent bone and even the extracranial tissues as well. Diagrams of the various ways in which the cranium may be affected were given in my Cavendish Lecture, and a good example of how the tumor may extend through the bone without destroying it is shown by a patient who has just been discharged and who therefore does not properly belong in this series of present house cases. Nevertheless, I wish to present the patient and show his tumor as an example of total removal.

Mr. G., 59 years of age, entered the hospital April 2 and was discharged April 29. Meanwhile he had this tumor removed in a single session under local anesthesia. He stood the three hour procedure well. There was no loss of blood; no change in pulse rate. The growth (Fig. 17), a 110 gram meningioma, is intact; it has been cleanly shelled out of the brain, and consists of a small extracranial and a larger intracranial portion. Beyond the disk of bone

*The photographs (omitted) of this case show the original tumor with its slightly torn surface and the recurrent 80 gm. tumor removed intact with dura attached.

Fig. 17.—Lateral view of parietal meningioma weighing 110 gm., extruding through (above) apparently intact skull.

Fig. 18.—Parietal tumor caused by extracranial extension of a meningioma.

there was one outlying intracranial nodule which might easily have been missed and which necessitated the rongeuring away of a further portion of the skull. This left a larger cranial defect than I like, but a considerable defect was necessary.

The photographs (Fig. 18) show his condition before operation. He was supposed to have a malignant tumor (sarcoma) of the skull. He had first noticed the "bump" on his right parietal region in August, 1922. A month later there was some disability of the left hand. He was treated for syphilis. Soon jacksonian attacks began in the hand. In time the leg became involved and then the face. A sensorimotor hemiparesis followed, most marked in the arm. The disk margins were only slightly hazy: there had been no severe headaches. The roentgen ray showed the skull to be intact though somewhat mottled at the situation of the tumor.

During the course of operation he appreciated the return of slight voluntary movement in the paralyzed hand. He made an excellent recovery and has already regained nearly full power of his arm: active movement and normal sensation returned before stereognostic perception was regained. It is still somewhat below normal.

The next example of these lesions presented a far more difficult diagnostic problem than either of the foregoing cases. It is difficult to realize when you see her that she still carries a large inoperable meningioma and was operated on only twelve days ago.

Case 20 (Walking).—Mrs. T., entered the hospital on May 6, complaining of "pounding headaches" and numbness of the right face. She is 34 years old and had perfect health until her present trouble which began eight months ago with what she describes as "sinking spells." These were vague attacks with gastric sensations, nausea and drooling, which occurred many times a day. Five months ago she first became aware of some haziness of vision for which glasses were prescribed. Since then vision has been progressively failing so that two months ago she was no longer able to read. Headaches did not begin until six weeks ago; they have been severe and usually nocturnal. At about the same time she first noticed some numbness of the right upper lip and gum. Two weeks ago diplopia was first observed, and latterly she has had some instability in walking, with tendency to fall to the left. She has lost a good deal of weight.

Examination showed advanced choked disk of 5 diopters; definite hypesthesia of the three divisions of the right trigeminal nerve; a poorly sustained nystagmus to right and left; inability to stand with eyes closed and a good deal of unsteadiness during progression.

It was difficult to tell from these symptoms whether her trigeminus was implicated by a growth above or below the tentorium, but the cerebellar symptoms were sufficiently definite to favor a posterior lesion and I think if we did not follow a fixed routine for all tumor cases and had not taken her fields of vision we might easily have been misled into a cerebellar exploration. Her fields of vision, however, showed a definite upper left homonymous notch (Fig. 19) which we have come to regard as one of the most significant signs of a temporal lobe tumor. This matter I discussed before this Association two years ago.

The roentgen rays, beyond evidence of increased intracranial pressure, showed nothing of localizing value.

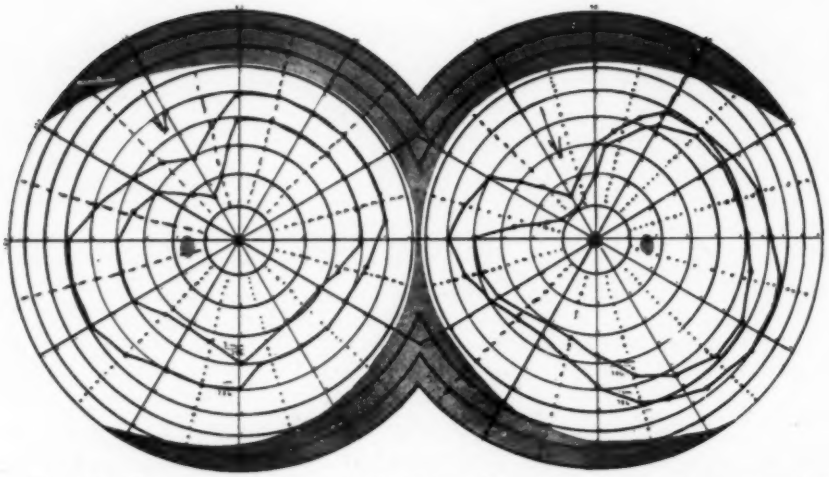

Fig. 19 (Case 20).—Fields on admission, May 12, 1923, showing upper left homonymous notch suggesting right temporal lesion. V.O.S., 20/40; V.O.D., 20/50.

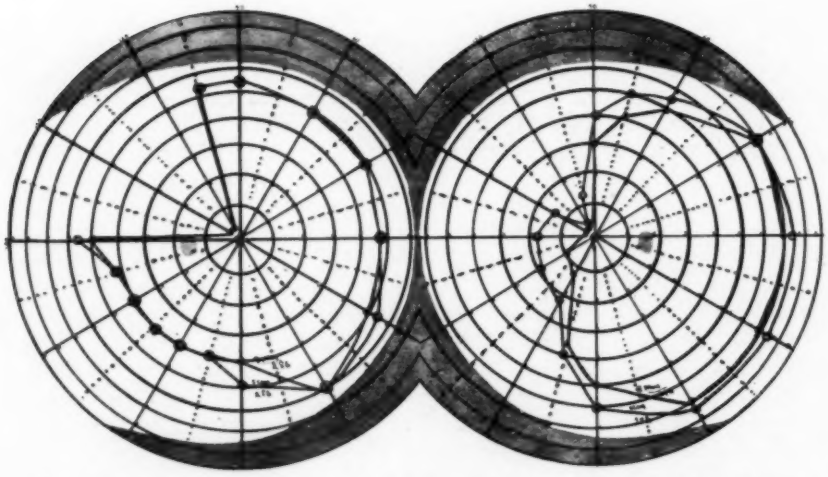

Fig. 20 (Case 20).—Fields on discharge, May 30, 1923, showing advance of constriction. V.O.S., 20/30; V.O.D., 20/40.

Before the notch had been detected in her visual fields, two of my associates had committed themselves on a diagnosis of cerebellar tumor, and I mention this to show how misleading the cerebellar signs may be in the presence of a temporal lobe lesion. In view of this difference of opinion, Dr. Horrax attempted a ventriculogram, air being introduced into the left occipital horn. He was able to secure only 10 c.c. of fluid, which was enough to show there was no hydrocephalus and therefore that the tumor must be supratentorial. The small amount of air which could be safely introduced did not pass from the left into the right ventricle. For several days she was very much upset by this procedure.

A right osteoplastic exploration was performed on May 18. The temporal lobe was bulging and its convolutions flattened. An incision carried through the second convolution came down on the smooth surface of what was taken to be an extremely vascular meningioma, so vascular that I did not venture to attempt its removal. A small fragment was taken for histologic verification,

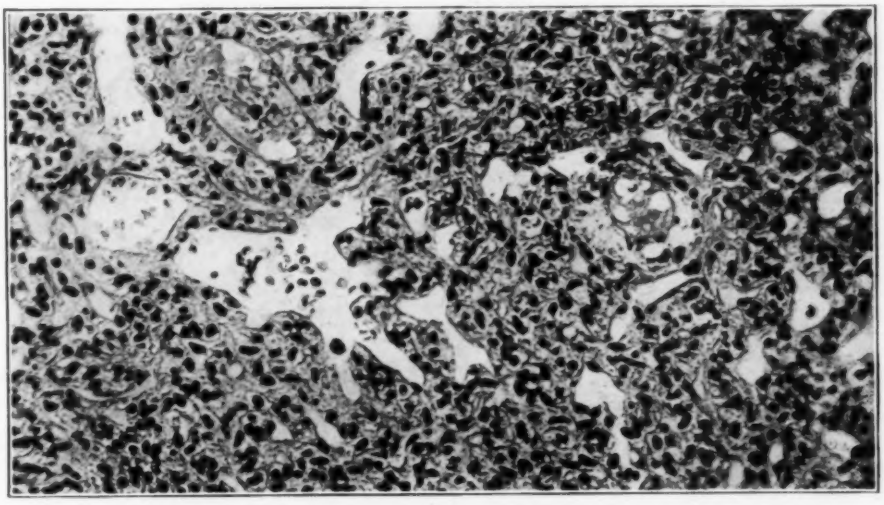

Fig. 21 (Case 20).—Section of tumor; diagnosis perithelioma. × 300.

and bleeding was so excessive that muscle had to be taken from the patient's leg and implanted before it could be controlled. An immediate subtemporal decompression was made and the bone flap replaced.

She has made an excellent recovery from the operation; her choked disks have subsided rapidly, and are now nearly flat, with restoration of reading vision. The fields, however, as would be expected, show advancing left homonymous constriction (Fig. 20) because the temporal loop has become further impaired by the outward dislodgment of the growth.

The chief interest in this case lies in the localizing diagnosis. A temporal lobe lesion may give a pseudocerebellar symptomatology which is very deceptive. Curiously enough since the operation her trigeminal numbness has entirely disappeared, yet I think we may assume that the growth has arisen from the temporal meninges overlying the ganglion.

How long her improvement will last I cannot foretell. I think she should have roentgen-ray treatments, though they offer less promise than in some of the gliomas and in the pituitary adenomas.[13]

THE NEURINOMAS

Tumors of the cerebellopontile angle have been long known to have a distinctive symptomatology. That most of them arise from the acoustic nerve has of late begun to be generally appreciated. In a monograph on the subject written six years ago it was pointed out that the histologic diagnoses returned by pathologists from the tissues of my early cases had been most variable — neuroglioma, fibroglioma, endothelioma, neuroma, fibrosarcoma, etc. It was only when the material from a large group of cases was finally assembled that the nature of the lesion was fully understood and the importance of the chronology of symptoms appreciated. Now we operate on these conditions in the majority of cases with full knowledge beforehand as to what we will encounter. It is a great advantage, particularly since there is possibly no more difficult and dangerous intracranial operation we may be called on to perform.

There have been seventy-seven of these cases in the series up to May 1. The seventy-eighth example is a patient sent here by Dr. Abrahamson. She is an intelligent Jewish woman and gives a good history of her malady. If she is correct in the chronology of her symptoms, however, her case is an exception to the usual rule of primary acoustic symptoms.

Case 21 (Bed case).—Miss S., is a school teacher, 26 years of age. Beginning nine months ago, she developed symptoms in the following order: (1) numbness of the lips and tongue on the right side; (2) difficulty in balancing and a deviation in gait; (3) buzzing in the right ear with impairment of hearing; (4) some obscuration of vision. These symptoms persisted with slight change until six weeks before admission when she began having diplopia. There has been practically no headache and only one attack of vomiting.

Examination showed a low grade of choked disk; complete right nerve deafness; marked horizontal nystagmus, slower to the right; a positive Romberg sign with typical cerebellar gait; considerable incoordination particularly of the right arm and leg. There was also loss of the right palatal reflex and some dysarthria. Caloric tests gave negative responses from the right ear.

The presumptive diagnosis of an acoustic tumor was made, though a recess meningioma could not be excluded. She gave an unusual chronology of

13. *Later Note.*—The tumor is found to be one of the rarer types of endothelioma—a so-called perivascular endothelioma (perithelioma) (Fig. 21), which is supposed to originate from the vessels of the leptomeninges and is unlike the meningiomas proper, which are of mesothelial rather than of endothelial origin. From its extreme vascularity, an attempt to remove the tumor, even had it been more accessible, would probably have been fatal. It is curious that there should have been two of these rare tumors (Cf. Case 27) in this short series of thirty cases.

symptoms. However, deafness of the left ear is more often noticed than of the right became of the use of that ear in the telephone, and her deafness, it may be observed, was first called to her attention by a physician. She has been studied by at least fifteen neurologists, otologists, and ophthalmologists.

Her operation was performed on May 15, and she is just beginning to sit up. The growth was thoroughly gutted until its capsule completely collapsed, 12 gm. of tumor in all being removed (Fig. 22). It was a fairly thorough intracapsular enucleation of a small neurinoma and I hope will serve to give her long and possibly permanent relief.

It is always difficult to know just how much to do in cases of this kind. Occasionally when tumors have reached a large size and have caused more marked general pressure symptoms than Miss S.

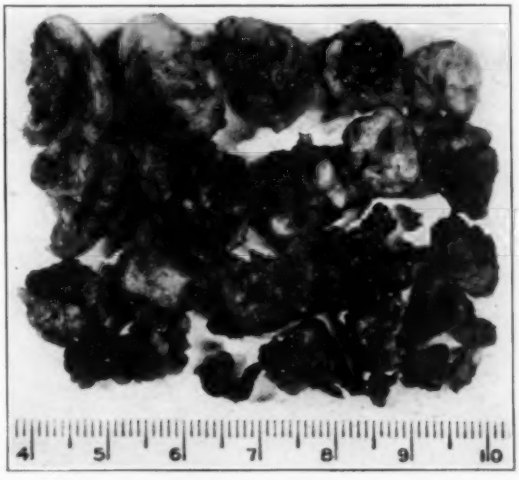

Fig. 22 (Case 21).—Tissue nodules (natural size) from intracapsular enucleation of neurinoma.

showed, a thorough intracapsular enucleation may not suffice to restore normal cerebrospinal fluid circulation and pressure symptoms may continue. Under these circumstances one may have to go in again and atttempt a complete enucleation, but this is extremely hazardous and permanent facial paralysis is almost inevitable.

One of course would always wish to do what Dr. Dandy has advocated in a recent note in the Johns Hopkins Hospital Bulletin: namely, carry out a complete extirpation. It, however, is exceedingly dangerous and I presume it is because of such attempts that the mortality which has been reported from certain clinics is appallingly great—twelve postoperative deaths in a series of fourteen cases from one foreign clinic.

Of course one should always make as thorough an enucleation as circumstances permit and occasionally one may be so fortunate as to

find a partially cystic neurinoma; I have been able to remove such a growth in its entirety, sparing the facial nerve in only three instances. I may be overconservative in these matters, but I would rather keep my operative mortality reasonably low than take unnecessary chances with these cases.

The next patient is the seventy-seventh case in the series, a man who was discharged just a month ago after a very thorough intracapsular enucleation of his tumor. He is already back at his trade.

Mr. B., is a loom-fixer, aged 25. He came here first in February, 1923, with a typical history of left acoustic tumor of seven months' duration—a blowing tinnitus followed by deafness; severe headaches increasing in severity; blurring of vision for six weeks; nystagmus, and unsteadiness in gait.

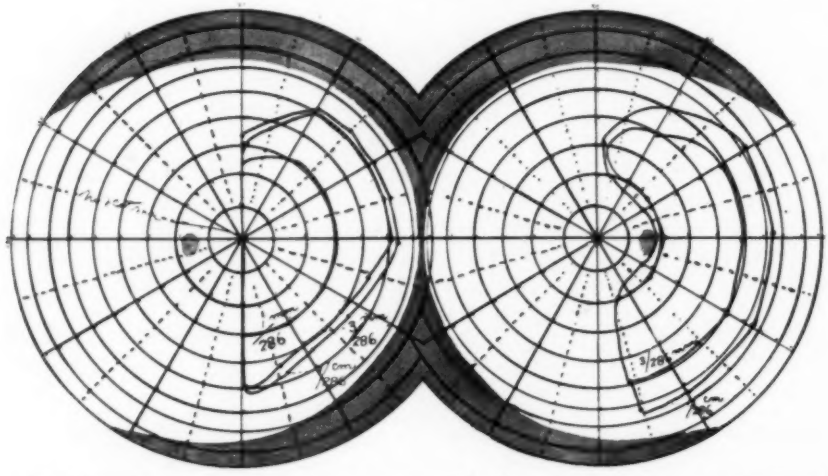

Fig. 23 (Case 22).—Left homonymous hemianopia from craniopharyngeal pouch cyst. V.O.S., 20/40 —; V.O.D., 20/70.

Unfortunately he had a bad furunculosis of the neck and it was necessary to postpone operation until the boils could be cured; it was a somewhat slow process.

It was not until March 15 that it seemed safe to operate. Meanwhile his choked disk had gone up from 2 to 4 diopters, and a definite hyperesthesia of the left cheek had appeared.

At the operation a partially cystic tumor was found in the recess, the gross appearance of which for a time led me to think it might be a tumor of the choroid plexus. Histologic examination, however, shows it to be a typical acoustic neurinoma. A fairly thorough enucleation was performed—so thorough that I fully expected him to have a subsequent facial paralysis, but he fortunately has escaped from this accident.

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14. Our figures for the Brigham Hospital cases, which gave a 13 per cent. mortality at the time of my last report (Further Concerning the Acoustic Neuromas, The Laryngoscope **31**:209, 1921) have crept up again, the operative mortality now being 13.5 per cent. for the series, which is none too good.

I started his operation under novocaine but it was too much for him and recourse had to be taken to ether. This, however, was withdrawn before the final closure, and during the last stages of the procedure he became restless and must have pulled out some of the deep muscle sutures. At the time of his first dressing it was found that there was a collection of cerebrospinal fluid under the scalp which necessitated a secondary repair of the wound, which accounts for his somewhat long postoperative sojourn in the hospital of nearly six weeks.

As you see, he walks steadily, turns quickly, has no nystagmus and no facial palsy. The disks are flat and show only slight secondary atrophy: he is able to read fine print with ease. It is now four weeks since his discharge and he has been back at work for three weeks. It may be considered a very satisfactory result.

CONGENITAL TUMORS

We come to quite a different group of cases—the cystic tumors arising from Rathke's pouch. They are sometimes loosely called suprasellar cysts though they may at times occupy the distended sella itself as in one of the cases I shall present. They might well enough have been considered in connection with the pituitary tumors proper. There happen to be three verified examples of these craniopharyngeal pouch cysts in the hospital at present. Moreover one of the patients with the lesion as yet unverified (Case 11) may have a tumor of this same nature.

The first of the three verified cases was operated on only a week ago. She is a young woman of 19, but with an immature aspect which gives her the appearance of a child of 12 or 13. Yet her mature bearing and mentality belie her seeming age.

CASE 22 (Wheel-chair).—Jeannette R., recently entered the hospital complaining of headaches and threatened blindness. Because of these things she had to leave school a few months ago—a particular disappointment because she has without difficulty always led her classes since she first went to school at the age of 6. Until she was 14, she was considered a normal child, but since then she has grown little if any and has remained sexually immature. Five years ago she commenced having periodic frontal headaches and there was a period, for a year or two, of pronounced polyuria. Her present visual field defects (Fig. 23) of an homonymous nature were first noticed four months ago.

There was no question about the diagnosis—a typical example of Fröhlich's syndrome: tumor, with hypophysial insufficiency. Examination showed primary optic atrophy with what was taken to be slight superimposed edema; a moderately enlarged sella with the shadow of a small patch of calcification (Fig. 24); a basal metabolism of —23; an incomplete physical development.

A right transfrontal operation was performed twelve days ago. The cyst, which was opened and evacuated, and which underlay the chiasm, was largely intrasellar. It was a little more difficult of access than usual because it did not separate the optic nerves but dislocated the entire chiasm upward.

She was not done quite as well as these patients ordinarily do. There has been no such prompt widening of the fields of vision as is usual, though she

experiences a definite increase in visual acuity. Moreover, a few days after the operation she acquired a palsy of the left external rectus. I am unable fully to account for it. Nor can I explain by anything seen at the operation why she should have shown an homonymous rather than a bitemporal field defect.[15]

The next case shows a far better result, for which Dr. Horrax may be given the credit.

Case 23 (Walking).—William C., is a clerk, 25 years of age, though he looks much younger. Since he was 16 his physical development has been at a standstill, but he has matured mentally and graduated from high school six years ago with boys of his own age, standing well in his class.

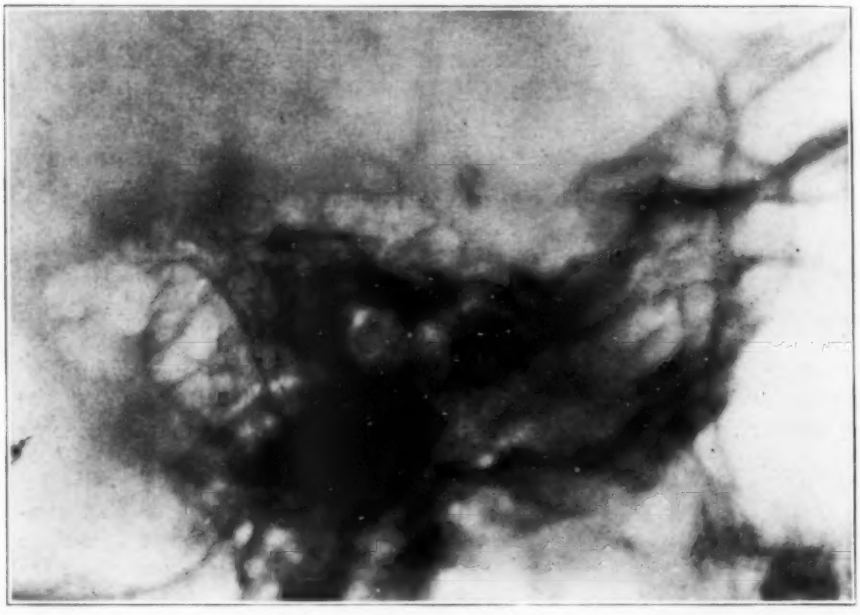

Fig. 24 (Case 22).—Deformed sella with shadow of patch of calcification in craniopharyngeal pouch cyst.

Three years ago he began having afternoon headaches, occasional vomiting, diplopia and blurring of vision in the right eye. He was treated for nephritis and later for anemia. He has taken pituitary extract for a long time. Two months before admission there occurred with some rapidity a marked loss of vision.

He has never shaved and looks like a boy of 16. He represents the lean rather than the fat type of dyspituitarism. Examination revealed a marked

15. *Subsequent Notes.*—Before discharge on June 15, she had begun to show marked and progressive improvement in her fields though the abducens palsy persisted. On July 23, she wrote that her eyes have straightened and her vision is "back to normalcy," which the laity at least can understand.

pallor of the optic disks; bitemporal constriction of the fields, more advanced in the right (Fig. 25); also a large sella with a few faint shadows of calcification above the posterior clinoids; a metabolism of —30.

Dr. Horrax operated on May 2, by the usual transfrontal route, found a suprasellar cyst containing the usual brownish fluid with cholesterin crystals.

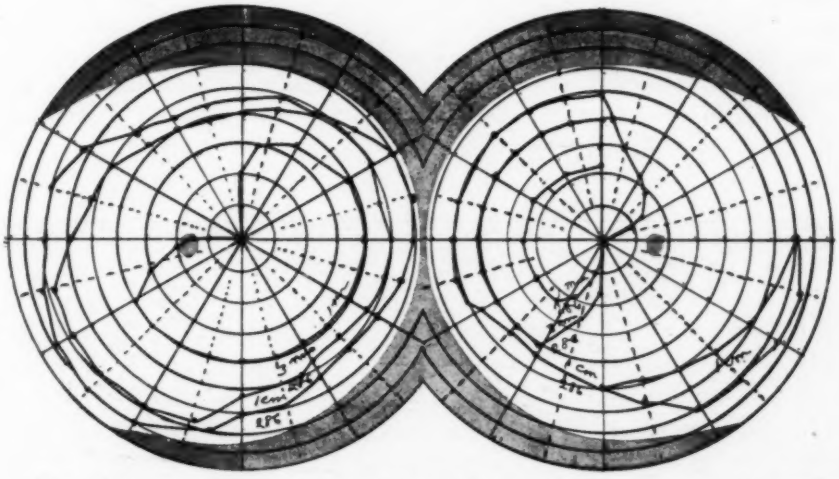

Fig. 25 (Case 23).—Fields of vision before operation in case of craniopharyngeal pouch cyst. V.O.S., 20/50 +; V.O.D., 20/50 +.

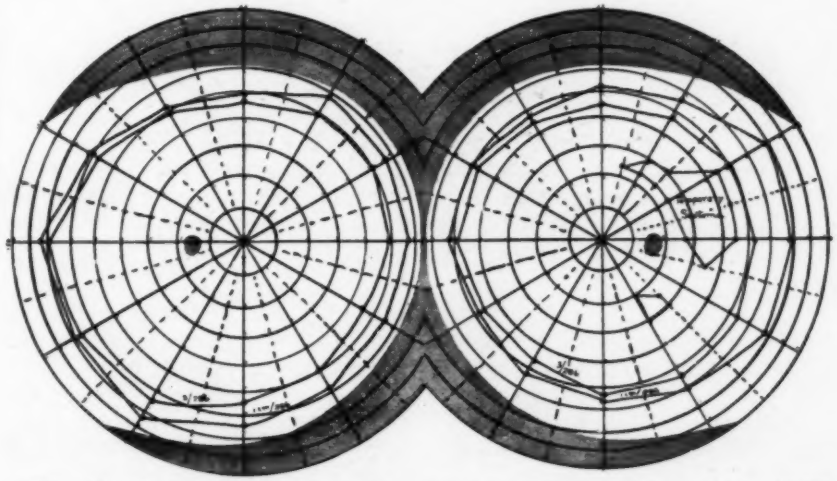

Fig. 26 (Case 23).—Fields of vision six days after operation. V.O.S., 20/20; V.O.D., 20/30.

A portion of the cyst wall was removed. It shows the typical mases of squamous epithelium characterizing these lesions.

There was immediate subjective improvement in vision the afternoon of the operation. The first postoperative fields were taken (Fig. 26) six days later (May 8) and showed normal field peripheries with a slight upper temporal scotoma in the right eye.

This is a good result. How the cyst will behave now that it has once been opened and drained is problematic. We can only hope that it will be quiescent for as long a time as has the cyst in the following case:

Case 24 (Walking).—This young man, now 30 years of age, was one of my Baltimore pituitary series. At that time, ten years ago, we were just beginning to get some glimmerings of light in regard to intrasellar versus suprasellar lesions, and I knew no better than to attack all cases from below, by the transsphenoidal route. In consequence, I nearly had a catastrophe in his case, for a pharyngeal pouch cyst occupied his fairly large sella, and with the collapse of the cyst a cerebrospinal rhinorrhea occurred and he had a sharp meningitis.

Fig. 27 (Case 24).—Sella showing shadows of calcareous deposits in wall of intrasellar pharyngeal pouch cyst and of 'clip' left on mucous membrane at time of transsphenoidal operation 10 years previously.

He entered the Johns Hopkins Hospital December, 1911, with the story of four years of severe cephalalgia and two years' gradual bitemporal loss of vision. He showed—and still shows (Fig. 27)—a large sella; a primary optic atrophy; and a bitemopral hemianopia with field defects which had advanced beyond a hemianopia (Fig. 28 *A*). Vision was reduced to the perception of shadows.

After the transsphenoidal operation his subjective improvement in vision was so marked that fields were taken the next morning, when it was found that normal peripheries had been regained on the left and a hemianopic field on

the right. Subsequently his meningitis developed and he had a close call, but made a good recovery with vision preserved.

Eighteen months later, May 30, 1913, he reported here at the Brigham Hospital and these fields (Fig. 28*B*) were taken by Dr. Walker. His vision then

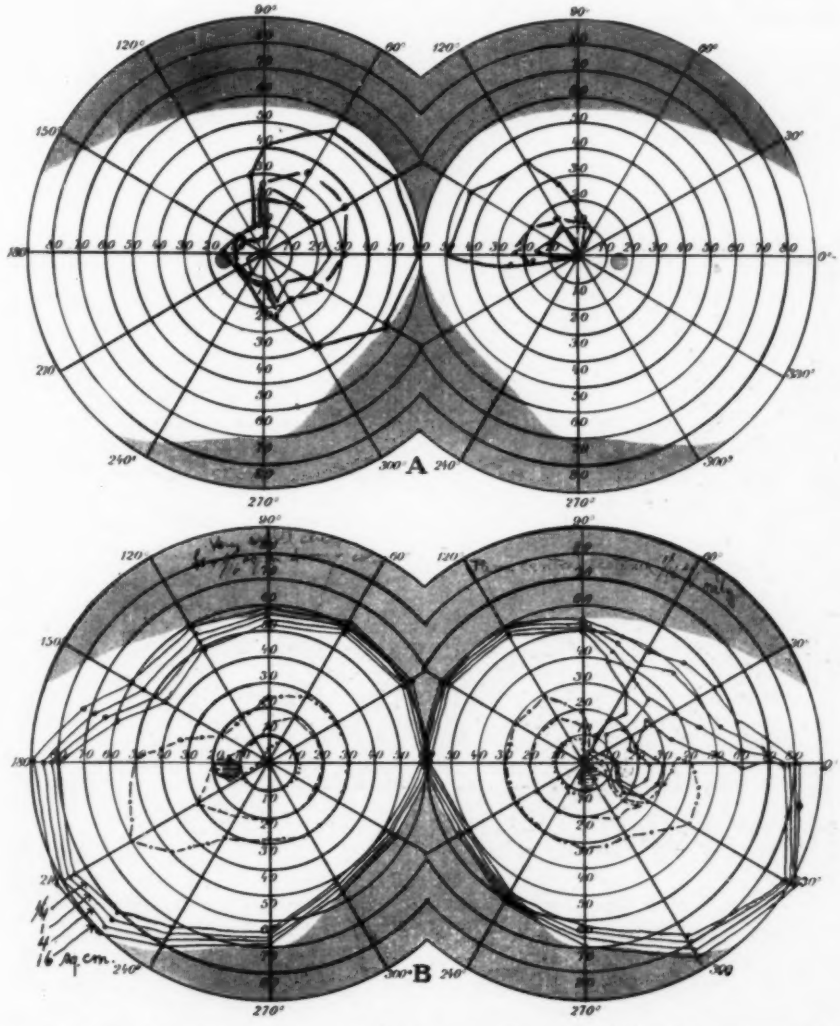

Fig. 28 (Case 24).—Fields of vision: *A:* Dec. 26, 1911, with 15 mm. disk, immediately before a transsphenoidal operation. V.O.D., 15/100. *B:* May 30, 1913, one and a half years after the evacuation of a pharyngeal pouch cyst. V.O.S., 20/30; V.O.D., 20/20.

was normal. For the next ten years I did not hear from him, but he remained well, and with normal fields (according to a series of charts which were taken) and normal vision.

A few months ago he first noticed a return of some temporal blurring. This progressed and he came here for advice on April 23. His fields showed a marked constriction and I was anxious about him, but not knowing for certain whether or not the trouble was progressing he was asked to report in a month. He has just returned. The fields show considerable advance (Fig. 29 *A*) and he is now no longer able to read newspaper print. He has had some double vision of late but practically no other symptoms—no discomforts whatsoever.

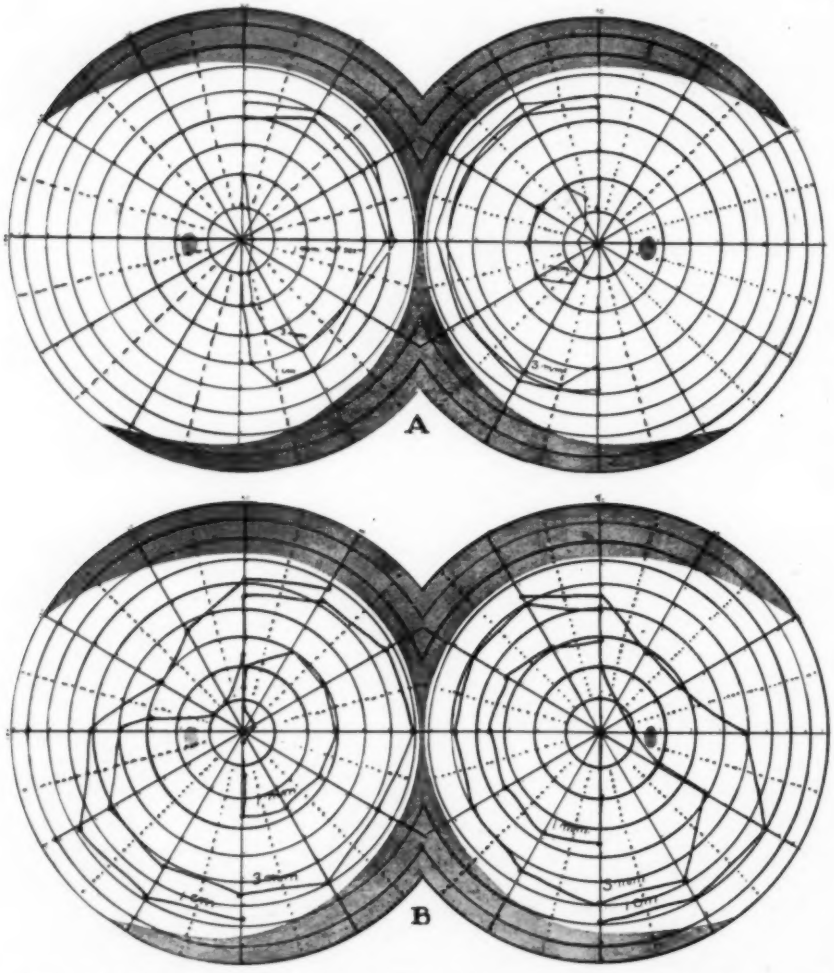

Fig. 29 (Case 24).—Fields of vision: *A:* June 9, 1923, just before second operation (transfrontal) after eleven years interval. V.O.S., 20/200; V.O.D., 20/100 +. *B:* June 20, 1923, six days after transfrontal operation. V.O.S., 20/70; V.O.D., 20/50.

The examination reveals merely a marked pallor of the nerves; a complete bitemporal field defect and a moderately enlarged sella within which the same calcareous deposits formerly observed are still to be detected (Fig. 27).

There can be no doubt but that he has a pharyngeal duct cyst whose calcareous portion largely occupies the sella. He ought to have a transfrontal operation—indeed, should have had one eleven years ago. In view of the fact that the cyst was merely emptied at that time, it is gratifying that he has had so long a period of unaffected vision.[16]

There are two other cases at present in the hospital that we assume have pharyngeal pouch cysts. Neither has been here very long, and their studies are not fully completed. I will show them briefly in connection with these three other cases, realizing fully that in the absence of suprasellar shadows one must speak with hesitancy of the pathology of these lesions whose site is so easily recognized. With Dr. Paul Martin I have recently published an article [17] on a series of primary chiasmal tumors which may be easily mistaken for the more common tumors of Rathke's pouch. I shall hope to say a word at the end of this session regarding a further case.

CASE 25.—This man, aged 32, sent here by Dr. Israel Strauss, entered the hospital last week. He has always had perfect health. He had a fine war record, and because of his exceptional vision was made a range finder in the navy. Twenty months ago he first noticed some blurring of vision. Subsequently there was diplopia and, for the past few weeks only, he has had severe frontal headaches which are chiefly nocturnal.

He shows nothing but bilateral primary optic atrophy and a clean cut bitemporal hemianopia. His sella is normal. Physically, to all intents and purposes, he is a healthy appearing man without pituitary manifestations.

What are we to do in a case of this kind? There must be something affecting his chiasm, and his best chance lies in the possibility that it may be a pharyngeal pouch cyst. The absence of shadows unfortunately makes this improbable.[18]

The next patient has an outspoken syndrome of pituitary insufficiency. He might be the twin brother of the young woman, the first of this short series. He also presents a typical Fröhlich's syndrome and has a feminine habitus.

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16. *Subsequent Note.*—He was not operated on until June 17, the vision at that time being reduced to 20/200 on the left and 20/100 on the right, and the fields (to a 3 mm. disk) showing a sharp bitemporal hemianopia. The left eye was blind to a 1 mm. disk. The upper cyst wall was removed together with a good deal of the calcareous material from the sella. There were no complications. He experienced immediate improvement in vision, and six days after the operation his fields had widened out to those shown in Figure 29 *B*. He was discharged July 4, in excellent condition.

17. Arch. Ophthal. **52**:209, 1923.

18.—*Subsequent Note.*—An exploratory operation on June 6 revealed a primary tumor of the chiasm—probably a glioma. No fragments were taken, and the case therefore remains one of "tumor unverified." He was given deep radiotherapy, though our experience is that this type of glioma is not benefited thereby. His condition on discharge June 21 remained practically unaltered. He died shortly after. No necropsy was performed.

CASE 26.—This young man, aged 18, has been in the hospital only a few days. He began three years ago to have severe headaches followed by periods of somnolence. Soon obscuration of vision was observed, primarily in the left eye. Subsequently the right became affected. He has had a period of polyuria, and during the past year has gained 25 pounds (11 kg.) in weight. Six months ago because of poor vision he gave up school. He is 5 feet (152 cm.) in height; his voice has not changed; he has never shaved.

Examination shows adiposogenital dystrophy; primary optic atrophy; partial bitemporal hemianopia (Fig. 30); a large sella (Fig. 31) with a suspicious suprasellar shadow; a basal metabolism of —8.

Were it not for the faint suprasellar shadow which suggests a pharyngeal pouch cyst I would be inclined to approach this boy's tumor through the nose, but I presume in view of our uncertainties as to the nature of the lesion it would be better to expose it from above.[19]

THE GRANULOMAS

Syphilomas of the brain are rare. I spoke in my address this morning of the view which was held not long ago regarding the propriety of a six weeks' antisyphilitic regimen for every case of suspected tumor. Horsley fought against this even in the days before the Wassermann reaction. Today there are probably few who still follow this dictum. One may nevertheless occasionally encounter a large fibrous syphiloma. I recently mistook such a tumor for a meningioma because of its gross characteristics and the way it adhered to the dura. The operative diagnosis, indeed, was "endothelioma" and it was not until the histologic report was returned that the true nature of the lesion was revealed. On the other hand I recently encountered and removed a tumor which I could have sworn was a syphiloma, but the microscope shows it to be an endothelioma. Not long ago, too, a syphiloma was mistaken for a tuberculoma until sections were cut. These experiences show how essential for a clinical verification the microscope may be,

19. —*Subsequent Note.*—At operation, June 9, the lesion proved to be a pituitary adenoma. A large portion of the gland, sufficient to free the distorted nerves from pressure, was scooped out from between the legs of the chiasm. Owing to the vascularity of the adenoma there was a good deal of difficulty in getting complete hemostasis before closure. His wound healing was not perfect because of a too tight bandage. Vision promptly improved, but he had marked postoperative exacerbation of his diabetes insipidus. This was controlled by intranasal pituitrin. He was discharged June 29 in excellent condition with his fields of vision practically normal, having improved from 20/100 to 20/50 in his left eye and from 20/50 to 20/20 in his right. He has subsequently done extremely well and his wave of polyuria has fortunately receded. It could have been avoided entirely, I believe, had his operation been a transsphenoidal one. The case, therefore, now classified as "pituitary adenoma: verified," was wrongly diagnosed because of a misleading series of roentgenograms. It will be seen that the preoperative pathologic diagnosis of each of these last two cases was wrong and that they do not belong with this group of craniopharyngeal pouch tumors.

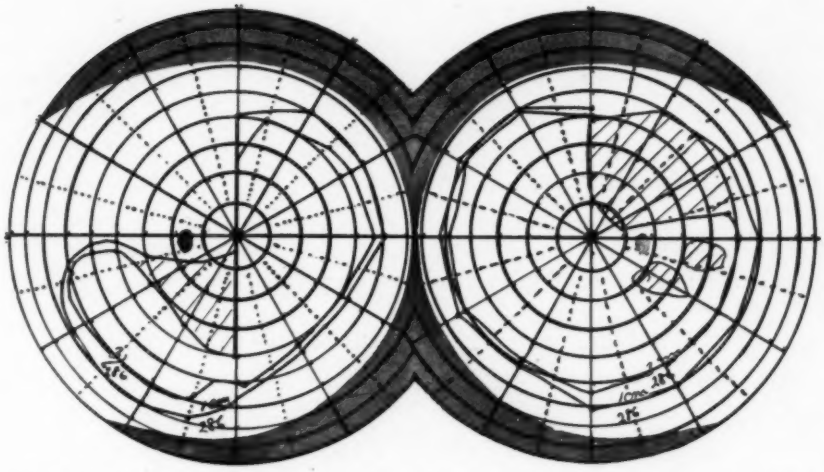

Fig. 30 (Case 26).—Fields of vision before operation, May 28, 1923. V.O.S., 20/100 + 1; V.O.D., 20/70.

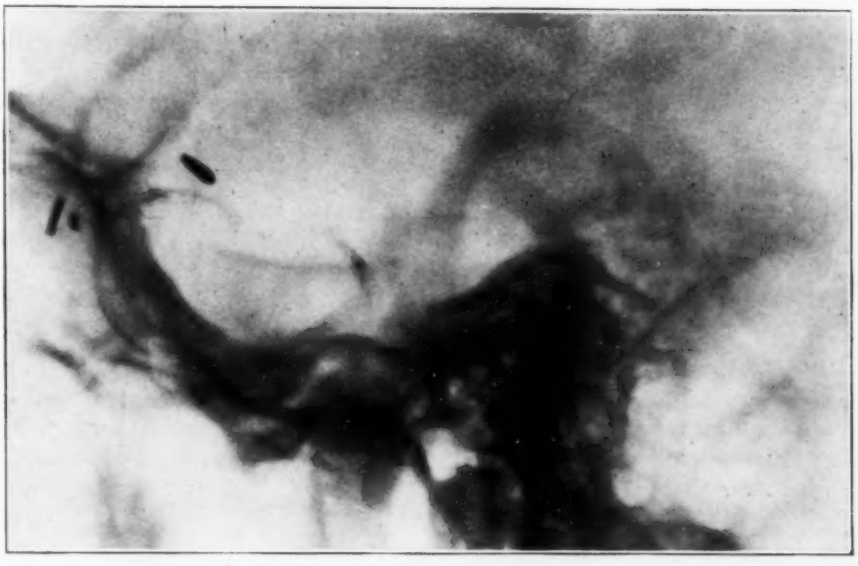

Fig. 31 (Case 26).—Sella turcica. A plate taken after operation showing one or two clips in place.

Tuberculomas also are rare, perhaps more so in an adult than in a children's clinic. My record with the surgical treatment of these lesions is bad. They may do very well for a time, but even after what appears to be complete enucleation, a tuberculous meningitis or a recurrence seems to be inevitable. My last case is now living six months after the removal of a large parasagittal tuberculoma, which before operation had been regarded as a presumptive endothelioma. Though every effort has been made to aid in her fight against the disease by the use of an Alpine lamp and attention to her general nutritive condition, it is evident from letters that a recurrence has already taken place.[20]

MISCELLANEOUS AND UNCLASSIFIED TUMORS

We can pass over with bare mention the metastatic and invasive tumors, the choroid plexus tumors, the angiomas and the group of unclassified or unclassifiable lesions. The metastatic growths are more common than the 3.9 per cent. of the verified tumors in the table would indicate, for being rarely subjected to operation most of them fall in the unverified group. Though one must be ever on the alert, they as a rule are easy of diagnosis and, generally speaking, an operation of any kind is futile. Nevertheless there have been some very extraordinary histories among the thirty-six cases—stories well worth telling though they do not properly concern this present clinic.

I have unwittingly during the past few months stumbled on two metastatic tumors. One of them, a melanoma, was removed from a woman's speech center, and almost before her hospital discharge multiple metastases appeared elsewhere and she survived only a few weeks. The other case I shall show tonight, even though he does not belong chronologically to this present group of patients. It is the sort of case, however, in which time counts, and it is now four months since his operation. His tumor is regarded by the pathologists as unquestionably metastatic, yet he seems to be thriving.

Mr. S., is a Jewish rabbi, 65 years old. Eighteen months ago, following an operation for femoral hernia, he had some temporary weakness of the left hand and leg which subsequently disappeared. Some months later (August, 1922) the same symptoms accompanied by paresthesia affected his right side, and speech became impaired. These symptoms rapidly progressed. He entered our medical service last December and was regarded as a case of cerebral arteriosclerosis. During the course of four weeks he became increasingly dull and his vocabulary became reduced to one or two monosyllables. He was too incooperative for a satisfactory neurologic examination. On January 3 a low grade of choked disk was observed and he was transferred to the surgical service.

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20. There was no suspicion that the tumor in Case 10, unverified at the time this meeting was held, might be a tuberculoma.

In view of a case with a similar syndrome we had carefully studied two years before,[21] it was recognized that he had a global aphasia with apraxia. The diagnosis of left supramarginal lesion was unquestioned: tumor probable: glioma most likely.

On January 16, under local anesthesia, this peculiar, nodular dark-colored growth (Fig. 32) weighing 51 gm. was carefully dislodged from his supramarginal gyrus. He improved rapidly. There was daily improvement in comprehension; in finding words, first in Yiddish; in making sentences; finally in conversation and in writing. His apraxia meawhile slowly disappeared. His hospital history has pages of voluminous notes on these matters. Dr. Bailey I hope will find time to report the case fully from a psychologic standpoint.[22]

Fig. 32 (Case 27).—Nodule of tumor dislodged from supramarginal gyrus.

This is all very satisfactory. But there is one "out" about the case. The pathologic report from a frozen section of a bit of the tumor taken at the operating table was returned "probable adenocarcinoma." It was subsequently reported to be a metastatic hypernephroma! Consequently he was gone over physically from top to toe—pyelograms taken, etc.—

21. Bremer, F.: Global Aphasia and Bilateral Apraxia Due to an Endothelioma Compressing the Gyrus Supramarginalis, Arch. Neurol. & Psychiat. **5**:663 (June) 1921.

22. Certain tests were made before the Association to see if there were any possible traces of the patient's former apraxia. He performed complicated acts promptly. Possibly the most interesting incident was his voluntary statement regarding his remembered confusion when he was asked to do similar acts during his convalescence.

but no primary lesion could be detected. By February 16, when he was discharged, he had made practically a complete recovery. I have not seen him till today, and, as you have observed he looks in perfect health.

GLIOMAS

We now come to what after all is the most important group and the least well understood—the gliomas, which supposedly represent about 40 per cent. of the tumors. A general idea prevails that these true tumors of the brain, rather than of the cerebral appendages, represent hopeless surgical lesions. As a matter of fact we know so little about them that we do not even have a satisfactory classification of their many varieties.

Dr. Bailey and I have set ourselves the difficult task of reviewing all the tissues in our large stock in the hope of at least separating out a few types from the main group as he has done with the ependymal lesions. For until we can begin to differentiate the gliomas, possibly on the basis of their gross appearances and certainly on the basis of their histogenesis, we are unable either to give a proper prognosis or to know what is the proper course to pursue when a tumor of one type or another has been exposed at operation.

Many of them, as we are all aware, are slow growing and may be carried for years; some may be enucleated without recurrence; some undergo complete cystic degeneration; some are susceptible to radiation; some if interfered with in any way whatsoever appear to grow with accelerated speed. But we have not the slightest idea what kinds of gliomas tend to behave in one way or another way.

In years gone by the best we could do was to subdivide the tumors on which a histologic diagnosis of glioma had been returned, into: (1) solid gliomas, (2) cystic gliomas, and (3) gliomatous cysts. This was done without regard for their histologic appearance, of which, indeed, I knew very little. It was enough for the time being to recognize that the cystic tumors were far more amenable to surgical treatment and therefore gave a better prognosis.

A year ago my assistant Dr. Martin made a thorough statistical study of all the cases recorded as "gliomatous cysts," then numbering ninety-five, and in his paper [23] makes an interesting comparison between the results of these operations and those for malignant disease of the breast. He shows that the expectation of life is greater after operations for this group of glioma cases than for cancer of the breast in spite of the operative mortality of 14 per cent. which in the case of the breast

23. Dr. Martin's paper, "La chirurgie des gliomes cavitaires" will shortly appear in the Archives Franco-Belges de chirurgie.

operations is practically nil. In short, 78.3 per cent. survived over three years; 46.3 per cent. may be considered as permanent cures; 6.7 per cent. had survived over the ten year limit.

But this admittedly is a most imperfect and crude classification and undoubtedly the 362 listed cases contain many tumors which further study will show to be capable of further subdivision; some of them, indeed, may prove not to be gliomas at all. We may recall that a few years ago many of the acoustic neurinomas were regarded as gliomas.

If all tumors arising from the primitive spongioblast are to be designated as gliomas, whatever their histologic characteristics, we need not trouble ourselves further. As a matter of fact, they need to be subdivided and there are various bases on which a subdivision can be made. Most pathologists bear heavily on the architectural arrangement of the cells, and thus the so-called neuroblastomas have come to be recognized as tumors without glia fibrils, which unquestionably arise from the spongioblast before its differentiation. But the name is misleading, for no one can possibly say that the cells in many of the tumors so designated are actually neuroblasts.

Undoubtedly we must seek other refinements than the architectural appearances alone, and the chemical affinities of the various cells in all probability hold the key. The school of Ramon y Cajal has shown that there are glia cells of several types, all showing special staining affinities. Apparently, the methods of the Spanish school have not been utilized in differentiating glial tumors, and steps certainly should be taken in this direction.

So far we have merely made a tentative subdivision of the tumors into the fibrillar and nonfibrillar or protoplasmic types. For the detection of the former Dr. Bailey has already perfected a stain far more simple and effective than Mallory's phosphotungstic stain so commonly used. In these directions we shall hope to progress.

Why, it may be asked, is all this of any concern to the neurosurgeon? It is of the greatest possible concern; for until we can learn to recognize and differentiate these tumors on a histogenic basis and learn much more about them than we now know, we cannot expect to differentiate them when exposed at operation, far less can we predict at the bedside what the lesion will prove to be. Hence until we can properly identify and classify these tumors we cannot know what operative procedure, if any, would best be carried out. It is only after we have taken this step that we can hope to answer the question as to why certain gliomas pursue such an unaccountably long clinical course; why certain ones are so amenable to surgical removal whereas others appear thereby to be merely stimulated into activity; why certain other types appear to be susceptible to a prolonged course of iodism or to deep radiotherapy and

others do not; and, finally, what types are to be expected in certain areas of the encephalon.

The first of these patients to follow will give an encouraging view of certain of these true brain tumors. The case is an unusual one and deserves a longer time than I can give to it. It evidences the help one may gain in obscure cases from a ventriculogram and brings up a question which has puzzled us greatly and which relates to the localizing value of astereognosis.

CASE 27 (Wheel-chair).—Mr. E., is 48 years of age, a merchant and of good habits. He has for years been the victim of severe headaches, but otherwise has had good health. A year ago he noticed that the left hand was getting clumsy: that he would drop things from it: that he could not use it to get small objects, like a piece of money, from his pocket. This symptom persisted. Six months ago his headaches became worse, were now occipital and extended down the right shoulder. There was occasional nausea and vomiting. At this time also he began to experience vertigo and some unsteadiness in gait. For some weeks before admission he had been bedridden owing to very marked instability. At the same time there had been very noticeable mental deterioration.

The outstanding features of his condition on admission over two months ago were: (1) static instability so marked that when put on his feet he would 'crumple up' unless supported, and (2) clean-cut astereognosis with loss of postural sense in his left fingers, hand, wrist and elbow.

We were at a loss to account for the syndrome, and the impression was gained that there might be two lesions: a right parietal and a cerebellar lesion. However, aside from some slight stiffness complained of on flexion of the neck, there was nothing besides the static instability to favor a cerebellar lesion. There was very low grade of choked disk. A few poorly sustained nystagmoid twitches could sometimes be elicited, but more often they were absent. There was considerable incoordination of the left hand, and the left arm showed some relative hypotonus, but this might have been due to a lesion in either situation. He had also definite hypesthesia to all forms of sensation in the left forearm. The deep reflexes were exaggerated with sustained clonus at the left ankle.

Had it not been for the astereognosis, which though now fading since his operation is still easily demonstrable, we would doubtless have felt, even in the absence of nystagmus, that he had a cerebellar tumor and would have operated for it without further ado.

All of us independently went over the case. Our findings agreed, though our interpretations did not. It was my own opinion that first or last we would have to explore the right parietal region and I felt that this would better be done first. Consequently, under novocain a flap was turned down, disclosing a bulging wet brain with hydrocephalus, but no lesion whatsoever of the cortex.

At the time of operation air was injected and the ventriculograms showed symmetrical dilatation of both ventricles. This made it almost certain that the lesion was subtentorial and in the absence of nystagmus I anticipated finding, if anything, a median lesion.

A cerebellar exploration was performed a month ago. It disclosed in the right hemisphere a large cyst whose contents were yellowish and clotted on standing. The cyst extended well down into the right cerebellar tonsil, and on opening it a surface nodule of tumor was disclosed which together with the

herniated tonsil projected down into the canal through the foramen. This was dislodged and the flat nodule measuring about 2 by 2 by 1 cm. was excised. It was intact so far as could be told.

It was a long, four hour procedure, but he has made a rapid convalescence. In such a fortunate case as this, the recovery, which should be permanent, is nothing short of a resurrection. A bedridden, disoriented, untidy creature has been transformed into a cheerful cooperative man who is able to get about alone. Fortunately vision has not been impaired.

In this case there are two things deserving of special comment. One of them, the enucleation of the tumor nodule, is possibly a purely surgical matter; but the other, which concerns his astereognosis, will be of general interest. I shall dismiss the first by saying merely that there have been several other examples of this procedure in the series—

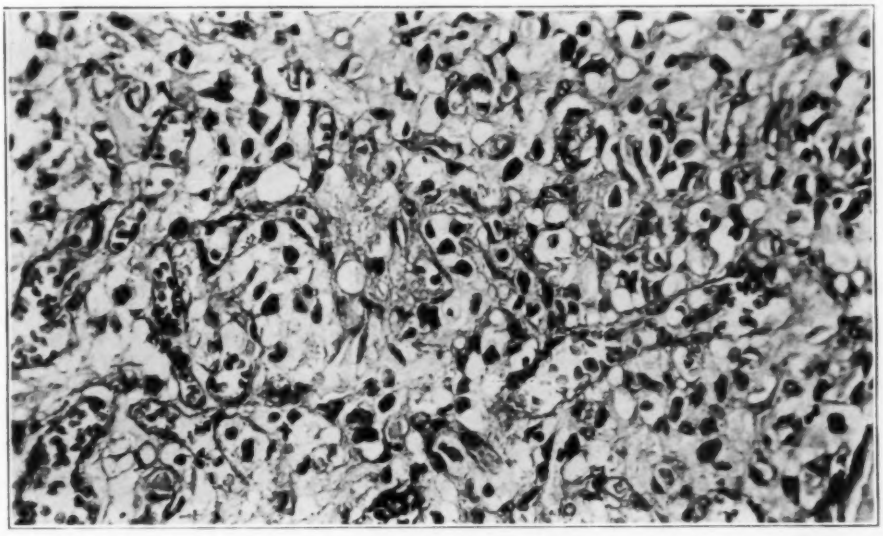

Fig. 33 (Case 27).—Section of cerebellar tumor regarded as a perithelioma. × 300.

that is, large supposedly gliomatous cysts from whose walls it has been possible to enucleate a single remaining tumor nodule. All of these cases have done exceedingly well. One of them at the present moment is a nurse in our training school; another is a patient from my Baltimore series who was operated on thirteen years ago and, though the gliomatous nodule showed cells undergoing active mitosis, he remains to this day well in every respect except for slight stationary impairment of vision; for he had at the time of operation an advanced stage of choked disk.

The other point of interest concerns Mr. E. H.'s astereognosis which made us suspect a parietal lobe lesion. This, I may say, persists and

though the impaired sense of position of the hand and fingers, with slight loss of common sensation, has been completely regained, he still has what we regard as an absolutely pure astereognosis. We have seen a number of patients with a comparable syndrome and shall hereafter regard astereognosis as a symptom that may accompany a tumor in the posterior portion of the cerebellum, which can in some way involve the sensory pathway.

I have dwelt on this case at some length because it is a good example of the struggles in which we may have to engage if we are successfully to pull through a patient with an obscurely localized lesion. It takes persistence and some courage, but one need never give up hope of doing something to relieve a person with a brain tumor.[24]

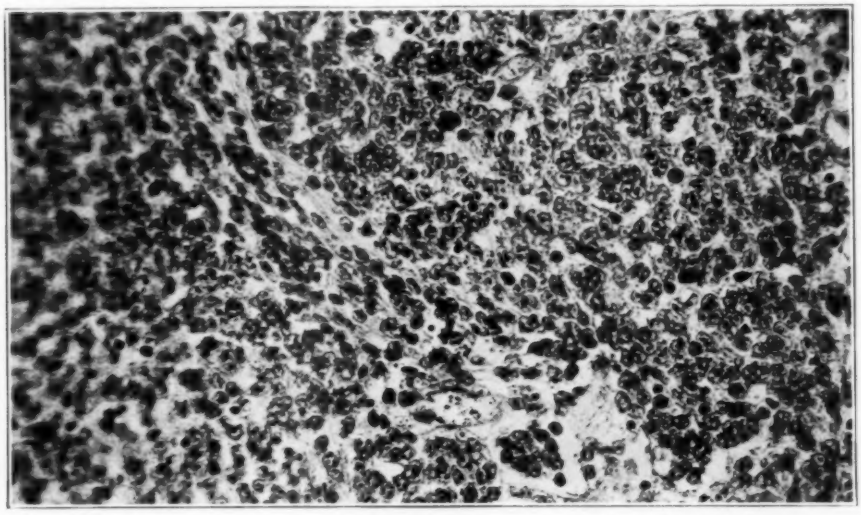

Fig. 34 (Case 28).—Section of neuroblastoma of fourth ventricle. × 300.

Four patients with verified glioma remain to be shown. The first is in an infant. It is difficult to do much in the way of a neurologic examination with a fretful and incooperative child who rebels even at the proximity necessary for an ophthalmologic examination. But this is quite a different child from the one that entered here on May 9.

24. *Later Note.*—The tumor of this case had been reported by the pathologists as the time of the meeting to be a glioma and they still adhere to this diagnosis. It, however, is a peculiar tumor which Dr. Bailey and I are inclined to believe is a perithelioma similar to the tumor in Case 20, which at operation was taken to be a vascular meningioma. We make this diagnosis reluctantly for if it is correct we will have to modify considerably our stand in regard to the justification of recording diagnoses of *"glioma: verified"* on the character of the cyst contents alone. In our opinion, therefore, the case stands as "cerebellar tumor: verified; cystic perithelioma" and is not a glioma at all (Fig. 33).

CASE 28.—This baby is 2½ years of age. She is said to have had influenza in February, subsequently became irritable and began to have shrieking spells followed by vomiting. She became unsteady on her feet and finally unable to walk alone. She appeared to be blind, with widely dilated pupils and what seemed to be secondary optic atrophy; the fontanel was open and bulging; there was marked incoordination of movement of the extremities; no nystagmus was observed. The roentgen ray gave no help in the diagnosis but it was thought to be most probably a cerebellar lesion.

On May 16, Dr. Horrax operated and disclosed a large solid, smooth tumor occupying the middle portion of the cerebellar field.[25] A fragment taken for histologic verification shows a neuroblastoma, so-called (Fig. 34).

I need not dwell on these midline cerebellar tumors. They may sometimes offer diagnostic difficulties. The next case is a similar one, though in an older child.

CASE 29 (Way-bed).—Helen M., 14 years of age, entered the hospital just two weeks ago. For a year and a half she has been growing clumsy on her feet and at entrance could walk only with marked staggering. For four months there have been suboccipital headaches, vomiting, and some left tinnitus.

The examination showed a high grade of choked disk; nystagmus; and the marked incoordination typical of a cerebellar lesion with hypotonus of all extremities particularly of the left.

At the operation on May 22, the ventricle was punctured early because of tension. The cerebellum was exposed without disclosing any abnormalities aside from a marked pressure cone, it being impossible to withdraw the herniated cerebellar tonsils from the foramen magnum. Punctures of the hemispheres were negative. Nothing was found in the recesses. There was no asymmetry of the hemispheres. Acting on an intuition, a median incision was made through the uvula and came down on a midline subcortical tumor, a sufficient amount of which was removed to permit cerebellar tonsils to be withdrawn and separated. This made it possible to see the growth occupying the roof of the fourth ventricle.

She has made an excellent recovery; is now free from headaches; her choked disks are now down to 1 diopter, fortunately with retention of normal vision. She will have roentgen-ray treatments. The tumor proves to be a glioma of fibrillary type (Fig. 35).[26]

This girl is of course enormously improved, largely because of her decompression, but owing to the position of the lesion I fear that her obstructive hydrocephalus may return ere long. The growth proves to be a glioma of different type from that in the foregoing case but we are not in a position as yet to give a comparative prognosis. However, I think the neuroblastomas of this region, of which we have had many examples, have the more favorable outlook—as good in all probability

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25. *Subsequent Note.*—The child made an astonishingly good recovery with preservation of considerable vision. She received her first roentgen-ray treatment and was discharged June 24.

26. *Subsequent Note.*—She was discharged June 23, greatly improved, with her choked disks flat.

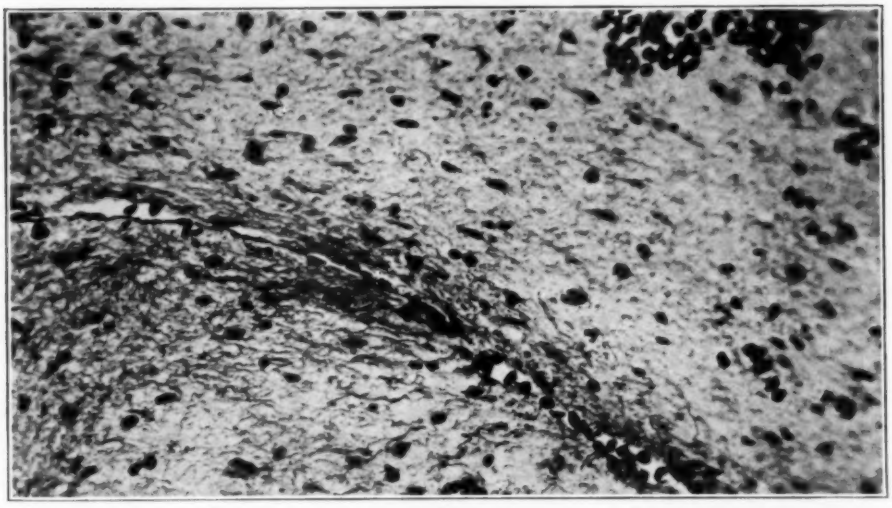

Fig. 35 (Case 29).—Section of fibrillary glioma from mid-cerebellar region. × 300.

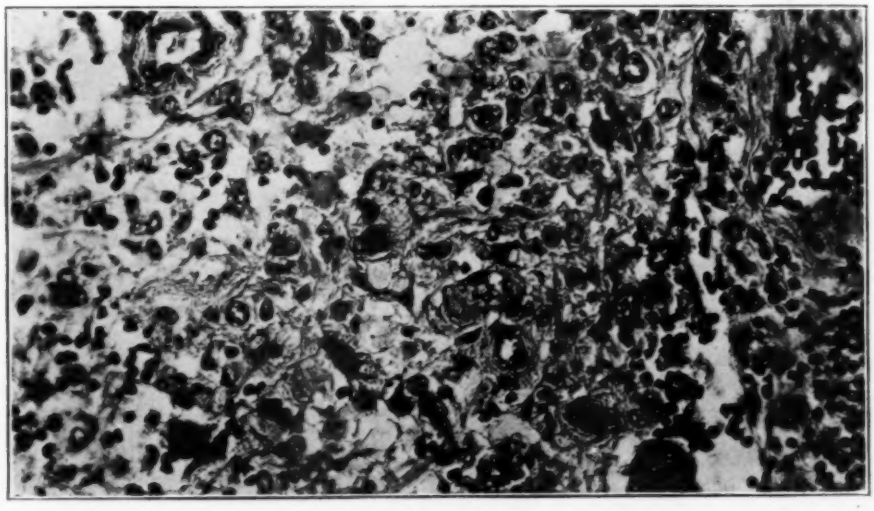

Fig. 36 (Case 30).—Section of right occipital tumor, a ganglioneuroma (?). × 300.

as the midcerebellar ependymomas; and Dr. Bailey this morning cited a case now living and in perfect health ten years after the tumor was exposed.

The next case illustrates what one may be sometimes forced into, namely the deliberate sacrifice of the bone flap in large inoperable tumors.

CASE 30 (Walking).—Mrs. H., is 50 years of age, and on May 10, entered the hospital, as so many of these patients do, because of failing vision and headaches. Eight years ago she had an abdominal operation, presumably for something other than malignant disease. Her inaugural symptom was loss of vision, and she has had some crude hallucinatory impressions (circles of light)—not the pictorial sort of hallucination discussed by Dr. Horrax this morning. She has had latterly, marked photophobia, severe headaches and vomiting, and for two weeks before entrance has been bedridden. She was thought to have encephalitis, and has been subjected before admission to a rigorous antisyphilitic regimen.

The examination showed merely bilateral choked disk of about 4 diopters, sharply-cut left homonymous hemianopia with bisection of the macula, and possibly some increase in the deep reflexes on the left.

There was little doubt in the minds of any of us but that she had a right occipital lobe tumor, and on May 21, Dr. Horrax operated and disclosed a large solid subcortical glioma with boundaries so ill defined as to render an attempted enucleation impossible. Consequently the bone flap was sacrificed and the scalp closed after a fragment was removed for study.

She has made an excellent recovery; headaches and vomiting have ceased; the choked disks have largely subsided. She will have roentgen-ray treatments and fortunately the lesion is in such a position as to cause no paralyses, so that she unquestionably may be able to resume her domestic duties for a time at least.

The tumor (Fig. 36) proves to be of an unusual type—one which has been described in the literature as a ganglioneuroma. It is made up of cells of the neuroepithelium which have differentiated imperfectly into both glia and nerve cells. It might be called a glioganglioneuroma or possibly a glioneuroblastoma. I do not know how effectively a growth of this kind can be checked by deep radiation. However, that is the best we can now offer. Occasionally under circumstances of this sort, and with the lesion in the right posterior hemisphere, one can without too much damage remove a huge glioma and give patients a long subsequent period of almost complete relief, with an abeyance of all symptoms except for the hemianopia (Cf. Case 12 of this series). Most neurosurgeons have doubtless had this experience, and some six months ago, in desperation over the outcome of a case very like that of this woman, I went in again shortly after a first stage ineffective operation, and removed a great ball of glioma the size of a fist; the man is now back at work and considers himself in perfect health.

The last of these glioma cases shows as good a result as the first of them—a similar surgical problem having been encountered.

CASE 31 (Wheel-chair).—Mary O'B., aged 20 years, a stenographer, entered the hospital April 20 with a typical history of tumor in the right paracentral region. She first noticed some disability of the left hand in November, 1922, which prevented her from typing. In January, the left thumb and forefinger became insensitive and numbness gradually spread to the hand and wrist. Soon the arm became increasingly weak, and finally totally paralyzed. Shortly before admission the leg and face also became involved. She has had attacks of severe pain radiating from the thumb and forefinger up the arm to the shoulder and trunk. She also had three jacksonian attacks ending in unconsciousness; also much headache and vomiting of late.

Examination showed choked disk of 3 diopters, left hemihypesthesia, and hemiparesis with slightly increased reflexes—they were difficult to obtain because of hypotonicity of the muscles. The arm was completely paralyzed and flail.

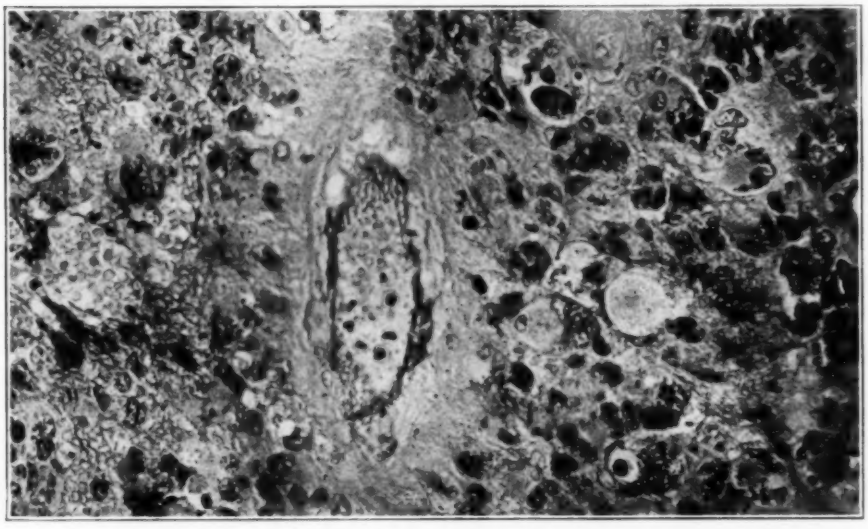

Fig. 37 (Case 31).—Section of nodule removed from the wall of a gliomatous cyst of the hemisphere. × 300.

April 24, an osteoplastic operation disclosed a large gliomatous cyst which was pointing in the prerolandic convolution about at the arm center. An incision was made into the cyst which, with the escape of a large amount of fluid, promptly collapsed so that satisfactory fixation of its walls was precluded. It was my impression that the cyst was tumor-lined throughout. At all events, there was tumor immediately adjacent to the cortical incision. Before replacing the flap a subtemporal decompression was made.

There was immediate marked improvement in motion and sensation, but contrary to expectation this was very transient and her paralyses soon returned as before. Her choked disks, after subsiding, again became swollen, with still higher measurements.

The cyst evidently had refilled and with misgivings five days ago (May 26) the lesion was reinvestigated. A more anterior cortical incision was made down to the cyst wall: 75 c.c. of fluid was withdrawn and immediately replaced by liq. formaldehydi until the walls were sufficiently fixed to hold their position

and permit of an examination. Two tumor nodules were thereby disclosed: one near the original incision and another near by. Both were removed in their entirety, so far as could be told.

From this second operation she has made an excellent recovery. Movement in the paralyzed arm was possible within a few hours after recovery from the anesthetic. She now, after five days, has almost completely lost the hypesthesia and is beginning to regain good power in arm and leg. The choked disks have already again receded to 2 diopters. The tumor (Fig. 37) is a glioma of peculiar type, difficult as yet for us to classify—an irregular cytoplasmic mass with numerous coarse fibrils, mitotic figures and large multinucleated cells. Diagnosis: "glioma: verified (type undetermined)."

This then ends our present May series of neurologic cases. It contains perhaps a more favorable group of verified gliomas than usual, but I am glad for this for I wish to leave an encouraging view in your minds regarding these lesions. Mary O'B., I hope and believe, has an excellent outlook, to judge from our other cases of a similar sort.[27]

FATALITIES

Out of a series of thirty odd cases of tumor one may expect with an average case mortality ranging about 15 per cent. that there will be at least two or three fatalities. The blow may yet fall on some of these patients as yet unoperated on who have so hopefully passed before us. Lest you may have gained the false impression, from the series of cases you have seen, that these tumor cases all do well, I must tell you of the month's experiences in the necropsy room, where the examination of six "brain tumor" cases is recorded—though only one of them, I am happy to say, the fifth, chances to be recorded as a postoperative fatality.

NECROPSY 1.—The first was a patient who was sent into the hospital in extremis with the history of a bad sinusitis. He died in a few hours. The necropsy showed a badly infected sphenoidal sinus which had involved a large pituitary adenoma that was almost completely destroyed by an abscess.

NECROPSY 2.—The second is the brain of an old glioma case operated on a year ago with partial tumor removal. The brain, removed after death and forwarded to us, shows a large right frontal glioma of protoplasmic type.

NECROPSY 3.—In the third case the brain also was sent from outside for study. The case was one of tumor suspect: chronic arachnoiditis. A suboccipital operation had been performed eighteen months ago with this presumptive diagnosis. There is no tumor and the necropsy therefore confirms the diagnosis.

NECROPSY 4.—The fourth case was a patient with a large right frontal glioma who entered the medical wards and died there without operation. She had a positive Wassermann reaction. The antemortem diagnosis lay between cerebral syphilis, encephalitis and tuberculous meningitis.

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27. *Subsequent Note.*—She continued to do extremely well. By June 6, she was up and able to get about the ward alone. Her choked disks had subsided without impairment of vision. She had received one roentgen-ray treatment, and was discharged June 18, walking from the hospital with hardly a perceptible limp.

Necropsy 5.—This brain, as yet uncut, shows on the surface a huge glioma in the left temporosphenoidal region. The patient was a man of 46, who had been under observation in a hospital with a diagnosis of encephalitis since February 20, when he had had a jacksonian convulsion beginning in the right side of the face. This was followed by aphasia, left sided headaches, hallucinations, and partial right hemiplegia. Choked disk was first observed on May 9, and in a few days he became exceedingly stuporous and was brought here in this condition.

He arrived in coma with Cheyne-Stokes respiration. Dr. McKenzie immediately gave him an intravenous injection of hypertonic saline solution and a few hours later he had so far regained consciousness that a fairly satisfactory neurologic examination was possible. It disclosed enough to justify the diagnosis of global aphasia and a probable lesion of the left supramarginal gyrus.

Under local anesthesia an immediate osteoplastic exploration with a generous decompression was made. This large inoperable glioma was exposed. The tumor lies in the Sylvian fissure and its extirpation, had it been possible, would have left him hemiplegic. It was the poor man's good fortune to die ten days later of a pneumonia.

Necropsy 6.—The last of these cases had a most unusual history, and we were never able during life to arrive at a satisfactory pathologic diagnosis, though the location of the lesion was fairly unmistakable. The patient was a school-teacher of 51, who was referred here last September from the Clifton Springs Sanitarium. Her trouble began in January, 1922, with headaches. In June, there was sudden diplopia due to a left abducens palsy. Subsequently the right abducens became affected, and left ptosis developed. There was marked photophobia. She had arterial hypertension, systolic pressure 200 mm. A roentgenogram showed a peculiar deformation of the sella with erosion of the posterior clinoid processes.

In addition there were evidences of hyperpituitarism, without acromegalic changes. She had a beard and had shaved for some years. Her metabolism was + 34 per cent. The fields were normal and the optic nerves, though pale, were regarded as within normal limits.

The marked degree of ophthalmoplegia without impairment of vision naturally suggested a pontile lesion, possibly vascular in view of the hypertension. However, the sellar destruction was definite and a transfrontal exploration was made. Lying below and behind the chiasm, the anterior end of a firm tumor, the nature of which was undetermined, was brought into view.

She was subsequently given deep radiation: the right eye regained its normal movements, and she was discharged.[28]

She returned last December for further roentgen-ray treatment. At that time she complained of a burning sensation in the right cheek. It was found that the right trigeminus had become involved and I was tempted to explore the subtemporal region on the supposition that she might have an endothelioma of the gasserian envelopes. Her oculomotor palsies were less marked than before and fortunately I procrastinated.

A month later the trigeminal anesthesia had disappeared and there was only a trace of the oculomotor palsies. A third roentgen-ray treatment was given.

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28. The case was reported by Dr. C. E. Case in the Clifton Medical Bulletin, 1923, p. 22, as "Pituitary Tumor Differentiated from Myasthenia Gravis."

By the end of February she began to have pain and stiffness in the neck and the trigeminal anesthesia reappeared.

On her reentry in March there was unilateral anosmia, beginning failure of vision, bilateral ptosis, some dysarthria and definite deviation of the tongue.

She returned again April 28. By that time the left facial, acoustic and hypoglossal nerves were involved. She was in great pain; deglutition and speech were difficult. The best we could do was to have recourse to euthanasia.

Fig. 38.—Adenocarcinoma of pituitary body spreading backward and bilaterally, involving all the cranial nerves before death.

There was an evident glandular metastasis in the left submental region; also rigidity of the neck.

Her release came only two days ago. Before that time, however, practically every cranial nerve from the olfactorii to the hypoglossi, so far as could be told on such examination as her condition justified, was either palsied or paralyzed on both sides.

The examination revealed an astonishing lesion which is feebly indicated by Miss Warner's sketch (Fig. 38). A flat hard tumor (adenocarcinoma)

extended from the pituitary region along the basilar process into the spinal canal, implicating, as can be seen, every cranial nerve. The growth had involved the sphenoidal body, the basilar process and the body of the atlas. There were multiple visceral metastases.

In these sorry ways we may sometimes verify obscure intracranial lesions. The diagnosis is "Tumor of the Pituitary Body, with multiple cranial nerve paralyses: Adenocarcinoma: Verified at necropsy."

CRURAL MONOPLEGIA AND PARAPLEGIA OF CORTICAL ORIGIN WITH A DISCUSSION OF THE CORTICAL CENTERS FOR THE RECTUM, BLADDER AND SEXUAL FUNCTIONS *

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PHILADELPHIA

Paralysis of an extremity or of part of one extremity may occur from a limited lesion of the cortex. Lesions of the internal capsule when present produce a more extensive paralysis, and it is almost inconceivable that a monoplegia or a segmental monoplegia could arise from a lesion anywhere in the cerebrum except in the cortex. Limited lesions of the cortex secondary to vascular disease may be so small as to produce aphasia, either motor or sensory, alexia, the parietal lobe syndrome or hemianopia, but it is unusual for a patient to have a monoplegia or a segmental monoplegia due to a vascular lesion; when such a condition exists it is perhaps more frequently seen in the upper than in the lower extremities.

Paralysis of one extremity or of a limited portion of one extremity is frequently seen in cerebral neoplasms, and was not uncommon during the late war in soldiers who had head injuries. The literature on cerebral monoplegia has recently been reviewed by Spiller,[1] to whose article the reader is referred for this information.

The first two cases presented are instances of crural monoplegia which were due to a limited vascular lesion, this being proved in the first case by necropsy. The artery involved in these cases is a branch of the anterior cerebral which supplies the upper part of the motor cortex and the paracentral lobule. This artery also supplies a rim of the cortex along the intercerebral fissue extending from the frontal pole to the occipital lobe and on the mesial aspect of the brain from the frontal lobe to the precuneus.

REPORT OF CASES

CASE 1.—*Clinical History.*—A white widow, aged 74, was admitted to the Philadelphia General Hospital, Oct. 26, 1920, in the service of Dr. W. G. Spiller through whose kindness I am permitted to report this case. Three

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* Read before the Forty-Ninth Annual Meeting of the American Neurological Association, Boston, June 1, 1923.

* From the neurological department of the School of Medicine, University of Pennsylvania and from the neurological service and laboratory of the Philadelphia General Hospital.

1. Spiller, W. G.: Contributions to Medical and Biological Research Dedicated to Sir William Osler **2**:1175, 1919.

weeks before admission she became suddenly weak in her right foot and was forced to go to bed; in the course of a few hours the weakness became paralysis and involved the entire right lower extremity. Her relatives thought that the condition was due to the excitement that followed a family quarrel. A short time after the palsy occurred, swelling was observed in the paralyzed part. The patient apparently had no spontaneous pain. The relatives knew nothing of her past medical history.

Physical Examination.—The woman lay in bed apparently suffering no pain. The right eye was missing, having been removed following an accident some years before. The left pupil was deformed because of an iridectomy; the pupil, however, reacted to light. No cranial nerve palsies were made out, although the patient was completely deaf and had been so for a number of years; the deafness came on slowly. Her speech, the upper extremities, the lungs, heart and abdomen presented no abnormalities. The right lower

Fig. 1.—Center of softening in the paracentral lobule; × 10.

extremity was completely paralyzed for motion and the extremity was swollen and edematous, its circumference being twice that of the left. It lay in a position of helpless eversion, a position frequently assumed in fracture of the hip. Manipulation of the right lower extremity caused the patient to cry out with pain, but the roentgenogram of the hip was negative for fracture and for evidence of arthritis. The deep reflexes in both lower extremities were present and normal. An abortive ankle clonus was present on the right and plantar stimulation produced a typical Babinski sign on the right and normal flexion on the left. So far as could be determined sensation was normal, although the sense of position could not be tested because of lack of cooperation on the part of the patient. There was no evidence of vertebral disease and the sphincter action was normal.

Examination of the blood, both chemically and for the Wassermann reaction, was negative. The urine showed evidence of a low grade nephritis, not incompatible with the state of her arteries and her age.

Clinical Course.—The paralysis in the right lower extremity improved to some extent, but the swelling remained constant. Jan. 12, 1921, she developed an acute gastro-intestinal condition and she died on the following day.

Pathologic Study of the Nervous System.—The brain showed a small area of softening (Fig. 1) involving part of the paracentral lobule and the extreme uppermost part of the motor cortex, and a small adjoining area of the superior tip of the parietal lobe; the softening was not sharply defined but blended into the surrounding tissue. The nerve cells in the surrounding tissue showed chromatolytic changes, and many gitter cells were seen. The meninges overlying the softening presented a reactive inflammation and the blood vessels within the area were the seat of an obliterating endarteritis (Fig. 2). The

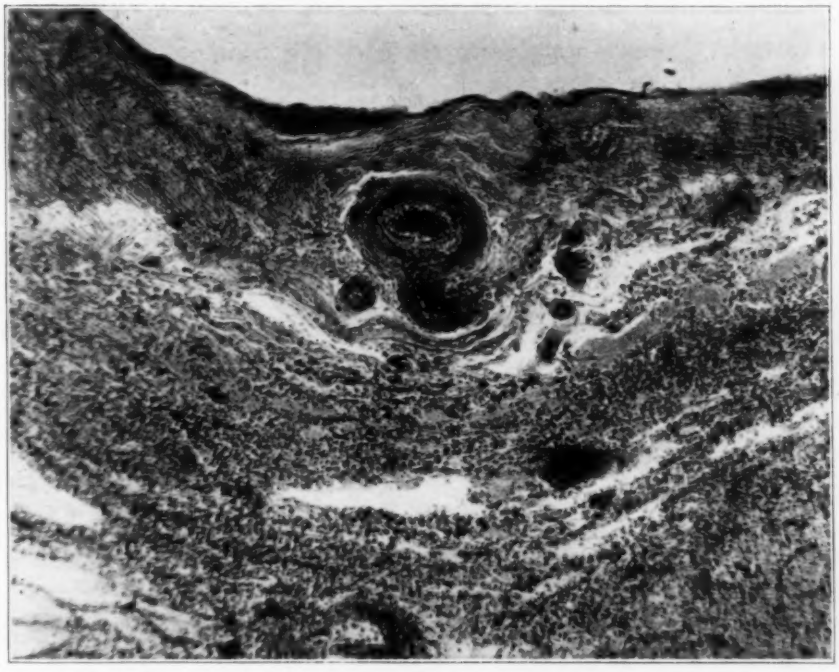

Fig. 2.—Area of softening showing obliterating endarteritis; × 115.

blood vessels throughout the brain showed intense sclerosis. The spinal cord showed a very slight degree of degeneration on either side of the posterior septum and the right pyramidal tract was the seat, as revealed by Weigert's stain, of a mild degree of involvement. With Marchi's stain the sections of the cord showed nothing abnormal.

The necropsy in general revealed a marked arteriosclerosis, lobular pneumonia, and an acute inflammation of the esophagus, stomach and the intestines.

Case 2.—*Clinical History.*—A white, married man, aged 54, was first seen at Salem, N. J., with Dr. R. M. A. Davis. The patient was admitted to the Hospital of the University of Pennsylvania Feb. 26, 1923, and died March 10, 1923. The past and personal histories were negative. His chief complaints were drowsiness and weakness of the right foot and leg.

In August, 1919, the entire left side of the body became numb and weak and the speech was thick; this cleared up in the course of the next two years. In January, 1922, he had a sudden attack in which the left side of his face twitched and his speech became a jumble of words; this passed off in a number of hours, but recurred the following day. After this he had what seems, from the description given by his wife, to have been a sensory aphasia which lasted several weeks and then improved, but the man was not as bright mentally as he had been. In January, 1923, he showed a tendency to fall asleep and slept a large part of each twenty-four hours. At this time he noticed weakness in the right foot which slowly involved the entire right lower extremity so that he dragged the foot when walking.

Physical Examination.—The man was drowsy during the first examination although at subsequent visits he was awake, but he took very little interest in the various tests to which he was subjected and he tired quickly. The station was normal and the gait was hemiplegic on the right. The cranial nerves were normal in every respect and the eye grounds showed no changes other than those of a moderately advanced arteriosclerosis. The upper extremities had good muscular power and showed no disturbance of sensation, coordination, or in the reflexes. Stereognosis was normal on both sides. The patellar reflex was active on the left and increased on the right. The left Achilles reflex was absent, the right was present but diminished, and ankle clonus was not obtained on either side. Plantar stimulation produced prompt flexion on the left and a typical extensor response on the right. All forms of sensation were normal in the lower extremities with the exception of the sense of position which was lost in the toes of the right foot. Hemianopia was not present. The man could read although he quickly tired and would not persist. He was not word deaf although at times very slow in complying with requests. The peripheral vessels were moderately sclerosed.

The urine, blood, kidney functional test, blood pressure and roentgenogram of the skull showed nothing abnormal. A lumbar puncture was not done because this had been performed at another hospital and was reported as yielding normal results.

Clinical Course.—March 4, 1923, the man became paralyzed on the entire right side and stuporous, and remained so until he died March 10, 1923.

COMMENT

These two cases represent instances of crural monoplegia due to thrombosis of a branch of the anterior cerebral artery. In the case of the woman with necropsy considerable doubt was present in the minds of some regarding the correct diagnosis; the case being considered by various physicians as one of phlebitis, fracture of the hip, myelitis and monoplegia due to a lesion of the cortex.

The diagnosis of phlebitis, while incorrect in this case, had some points in its favor. The sudden onset, the swelling and pain on motion, point in a measure, at least, to disease of the femoral vein. The absence of spontaneous pain and the presence of a typical Babinski sign ruled out this condition. Monoplegia may result from phlebitis, this condition having been present in two cases of brachial monoplegia which I reported as coming on after a phlebitis of the subclavian vein.

The position which the lower extremity assumed, that is, one of helpless eversion, and the fact that manipulation of the extremity produced pain suggested to the minds of some that the woman had a fracture of the hip, a condition which was ruled out by a negative roentgenogram and by the extensor plantar response.

The diagnosis of a spinal lesion as the cause of this woman's symptoms appeared far fetched because it is almost inconceivable that a lesion of the spinal cord in the lateral section could be so small as to produce nothing except a paralysis of one leg; one would certainly have expected, if this were the case, that at least a partial Brown-Sequard syndrome would have resulted.

The one remaining consideration in this case was that the woman had a monoplegia, cortical in origin, because any lesion as low as the capsule owing to the compactness of the fibers in this location could not produce a monoplegia or a segmental monoplegia.

Case 2, although without necropsy, was in all probability a similar condition. Because of the slowness of the onset, a tumor at one time was thought to be a likely diagnosis.

BILATERAL CORTICAL LESIONS

The remaining cases which form a basis for this report are instances of paralysis of both lower extemities with incontinence of urine and feces, and in one, paralysis of the sexual functions due to bilateral cortical lesions. Paralysis of both lower extremities due to a cerebral condition is uncommon and it will be recognized that the lesions in such a case must be bilateral.

Bilateral thrombosis of the arteries supplying the occipital lobe has been known to occur, but so far as I know there is no case on record in which there was a bilateral thrombosis of the arteries supplying the upper part of the motor cortex. It is possible that a tumor could involve both leg centers, but such an eventuality is only infrequently, if ever, seen.

The two other possibilities which could produce a cortical crural paraplegia are thrombosis of the longitudinal sinus and injury to the brain. The superior longitudinal sinus receives on either side the veins which drain the mesial aspect as well as those which carry blood from the upper lateral half of the surface of each hemisphere; the veins of the lower half of each hemisphere pass through the Sylvian system. A free anastomosis is often present between the two sets of lateral cerebral veins, and consequently complete occlusion of one set may not necessarily produce a permanent blocking of the venous outflow from the area naturally drained by it. A sudden blocking may abolish, at least temporarily, the functions of a part of the area normally drained by the venous system affected.

Holmes and Sargent [2] in their article on "Injury to the Superior Longitudinal Sinus" note sixteen cases in which only the lower limbs were affected, and thirty-one cases in which both legs and one arm were weak. All of their cases were due to head wounds.

REPORT OF CASES

CASE 3.—*Clinical History.*—A black married woman, aged 38, was admitted to the Philadelphia General Hospital Feb. 2, 1923, in the service of Dr. M. A. Burns to whom I am indebted for the privilege of using this case. She had had about fourteen pregnancies, ten of them resulting in miscarriages. She had four children living and well, the oldest being twenty-one years of age. The patient was married twice; her first husband died of paralysis, the second is living and well.

In the latter part of October, 1922, she became unconscious and fell down four steps. When she regained consciousness in a few hours she found herself sitting in a chair, and completely paralyzed from the waist down. She was also weak but not paralyzed in the right arm, and had a rather marked difficulty with her speech which persisted for one month and then cleared up. At the end of six weeks she could walk by pushing a chair in front of her. The weakness in the right upper extremity disappeared with the exception that she could not completely elevate the arm at the shoulder. From the time she regained consciousness after the onset she was entirely incontinent of urine and feces. For two months before admission she had been bedfast and had complained a great deal of pain in the right lower extremity. Both legs became swollen a month before admission to the Philadelphia Hospital. She had no headache and no trouble with the eyes or ears.

Clinical Course.—The condition remained unchanged until March 20, 1923, when she became stuporous, and on the following day weakness of the entire left side of the body was noted. March 23, 1923, she had three generalized convulsions and between these she had twitchings of the left side of the face, left arm and the left side of the abdomen. Her entire body became swollen and she died March 28, 1923. Her clinical chart showed a temperature which ranged from 99 to 101 degrees from the time of her admission until March 20, 1923, when it rose to 103, and the last two days of her life her temperature gradually mounted until it was 107 by axilla shortly before she died. Her pulse and respiration curves showed a rise proportionate to the fever.

Physical Examination.—The woman, who was obese, spoke clearly although she contradicted herself on a number of minor points; she gave a history of her illness rather accurately as checked up by the report of a relative. She lay in bed and cried out with pain in the legs whenever she was moved. She could neither turn over in bed nor sit up without assistance. The pupils were equal, large, regular, and reacted well to light and in accommodation. The cranial nerves were normal, although it is proper to state that the eye grounds were not examined. The upper extremities showed perfect function with the exception of limitation of motion in abduction of the right shoulder joint. When the right shoulder joint was passively moved the patient complained of great pain in this region, although a roentgenogram of the shoulder was negative. Coordination in the upper extremities was good.

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2. Holmes, Gordon, and Sargent, P. W. G.: Brit. M. J. **2**:493 (Oct. 2) 1915.

The lower extremities were completely paralyzed and were in a position of helpless eversion. Whenever either lower extremity was moved the patient complained bitterly of pain in the part moved, although she could not localize the pain. Both lower extremities were considerably swollen, the left more than the right. All the deep reflexes were normal, although it was difficult to obtain those in the lower extremities because of the edema. Plantar stimulation produced no movement of the toes on either side. Touch, pain, heat and cold were normal throughout and no note was made on the record concerning other forms of sensation. The woman was extremely obese and

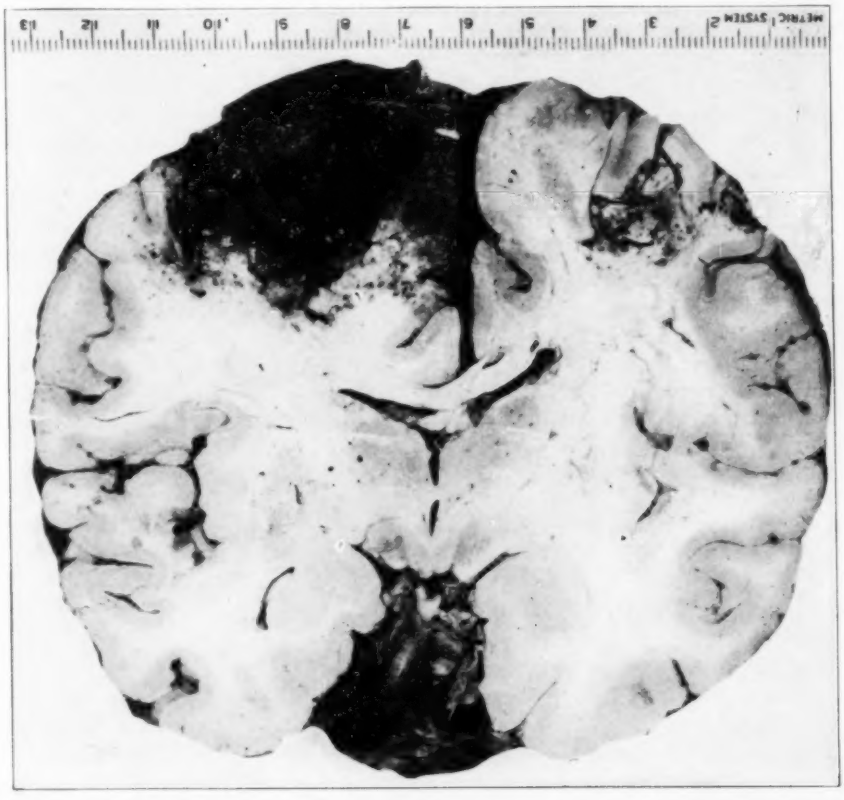

Fig. 3.—The brain of Case 3 showing hemorrhages occupying the upper part of both motor areas.

the fat on the abdomen hung in a large apron-fold over the pubes. A moderately large tumor was present in the right lower quadrant of the abdomen; this tumor was smooth and of rubbery consistency; catheterization did not reduce its size. A loud systolic murmur was present over the mitral area and was transmitted to the axilla. The pulse was regular and the blood pressure was 165 systolic, and 100 diastolic.

March 20, 1923, as noted above, the patient became completely paralyzed on the life side of the body, had three generalized convulsions and between the attacks she had twitchings of the left side of the face, left arm and left

side of the abdomen. Feb. 22, 1923, the urine showed a trace of albumin without casts, while on March 22, 1923, a heavy trace was present with a few hyaline casts. The blood urea was 19 mg., Feb. 12, 1923, and on March 15, 1923, was 15 mg. per 100 c.c. The blood sugar was 125 mg. March 12, 1923, the blood Wassermann was three plus with cholesterinized antigen and negative with the Noguchi antigen; March 15, 1923, the Wassermann reaction was two plus with the cholesterinized and negative with the Noguchi antigen. The spinal fluid was normal in every respect when examined March 15, 1923.

Pathologic Study.—The brain was small; the frontal lobes were long and narrow, and appeared hypoplastic. The meninges were thin over the right superior frontal cortex; a hemorrhage of large size, with considerable disturbance of the underlying tissue extending to the superior surface of the cortex, could be made out distinctly in this area. On sectioning the brain (Fig. 3), the upper part of the motor cortex was found to contain many minute hemorrhages on the left side, whereas on the right there was a large hemorrhage with softening; this area extended 5 cm. downward from the intercerebral fissure and 5 cm. into the subcortical region, although it did not involve the corpus callosum or lenticular nucleus. The hemorrhage on the right side of the brain extended from 2 to 3 cm. back of the rolandic fissure, and 6 to 8 cm. in front of it. On the left, the petechial hemorrhages were confined almost exclusively to the upper part of the motor area. Pial veins in the left frontal region and right precentral and frontal regions were thrombosed. The left paracentral lobule showed many microscopic areas of softening. A thrombosis of the superior longitudinal sinus was present just anterior to the rolandic fissure; the center of the thrombosis showed liquefaction necrosis, a thick reddish gray material being obtained on section. Smear of this material revealed a coccus. The spinal cord was removed only as far as the cervical region; in this part of the left crossed pyramidal tract showed a diffuse but mild degeneration.

The general necropsy showed chronic mitral disease, chronic diffuse nephritis, an infarct of the right lung and fibroids of the uterus.

Case 4.—*Clinical History.*—A white, unmarried man, aged 27, was first seen at the U. S. Veterans' Bureau on Sept. 30, 1921. His past and personal histories were negative with the exception of a head wound incurred in action. The patient denied venereal disease and the use of alcohol.

Aug. 11, 1917, while a private in the One Hundred and Eleventh Infantry, he suffered a severe head wound. He was unconscious for seven days and then remained between the lines for six more days before being picked up by the Germans who made him a prisoner of war. When he regained consciousness he was paralyzed for motion from the waist down, and he was unable to control the bowels or bladder. For some months he noticed that all sexual desire and power were absent. Dec. 1, 1918, he first noted a return of power in the right hip and shortly after this in the left hip; all through his convalescence the right lower extremity improved before the left. He was first able to stand about the middle of March, 1919, and began to walk shortly after April 1, 1919. Control of bowels and bladder and the return of sexual functions took place shortly after movement was noticed in the right hip.

From the time he regained consciousness he was certain that there was no loss of sensation in the lower extremities.

Physical Examination.—The man was fairly well developed and nourished, and presented no abnormality above the hips with the exception of a depression in the interparietal region. A roentgenogram of the skull by Dr. David

R. Bowen of the Pennsylvania Hospital revealed the following: "There is a large defect in the interparietal region just behind the coronal suture; this defect is roughly ovoid with a linear bone formation extending through it. The edges of the defect are clean cut and give every evidence of being permanently healed."

The gait was peculiar, the base of support being widened and the gait being a combination of spasticity and bilateral foot drop. The station was normal. The abdominal and cremasteric reflexes were present and normal. Plantar stimulation produced a slow extension of the great toe on both sides. The patellar and Achilles reflexes were exaggerated but ankle clonus was not obtained. Marked weakness was present in all movements of the lower extremities especially those of the feet where little motion was present. Sensation was normal in the lower extremities with the exception of a slight disturbance of the sense of position and of tactile localization in the toes of the left foot.

His ability to walk, as already mentioned, returned about April 1, 1919, although he could not walk any distance until the latter part of August, 1919. At present his lower extremities tire very easily. The return of control of bowels and bladder is complete and sexual functions are normal.

COMMENT

Cases 3 and 4 represent crural paraplegia of cortical origin, due in both instances, I believe, to involvement of the superior longitudinal sinus—in Case 3 to disease and in Case 4 to injury.

In Case 3 the primary cerebral insult resulted in paralysis of both lower extremities, weakness of the right upper extremity and some disturbance of speech. The weakness in the right upper extremity disappeared with the exception of the movements of the shoulder, and the disturbance of speech vanished entirely. She then presented a paralysis of both lower extremities with incontinence of urine and feces which persisted until death five months later. There was intense pain in both lower extremities whenever they were moved; a similar condition existed in Case 1 when the paralyzed extremity was manipulated. While neither of these patients had involvement of the thalamus, they did have pain, central in origin, which suggested thalamic disease.

Case 4 was one in which injury to the superior longitudinal sinus occurred following a head wound. In this man the paralysis of the bowels, bladder and sexual functions persisted at least four months, and the return of these functions occurred simultaneously with movements of the hips. It will be noted that in both of these cases incontinence of urine and feces occurred.

Grünbaum and Sherrington [3] in their experimental work on apes place the cortical centers for the rectal, bladder and sexual functions at the very top of the motor convolution, or in other words in the foot center. Czylharz and Marburg,[4] in at least two articles which they

3. Grünbaum and Sherrington, C. S.: Proc. Roy. Soc. **69**:206, 1902.

4. Czylharz and Marburg: Jahrb. f. Psychiat. 134, 1901; Wien. klin. Wchnschr., 788, 1902.

have written, place the cortical center for the bladder in the hip region and Friedman agrees with them.

Holmes and Sargent [2] mention a number of cases in which the functions of the bladder were affected. In the majority of these cases there was at first difficulty in passing urine or even retention, but this usually disappeared quickly. These writers say that incontinence occurs less frequently, although in a few of their patients it was present and persisted for a considerable length of time; the bladder apparently emptied itself reflexly when it had filled to a certain point.

SUMMARY AND CONCLUSIONS

The four cases here reported present unusual features. Case 1 and Case 2 are instances of crural monoplegia of cortical origin, proved in Case 1 by necropsy findings and due to vascular disease in Case 2. In Case 1, with complete paralysis of the right lower extremity, the patient had no disturbance of the sphincters, and although Case 2 was not paralyzed completely in the right lower extremity, he was very weak in this part and at no time did he present disturbance of the sphincters.

Cases 3 and 4 were paralyzed in the lower extremities and had incontinence of urine and feces. In Case 4, a male, there was paralysis of the sexual functions. Case 3 never regained ability to control the bowels and bladder. At necropsy the brain showed bilateral lesions involving the upper part of the motor convolutions. This woman had tremendous edema of both extremities, a condition which was also present in the paralyzed limb of Case 1.

Cases 1 and 3 also had intense pain in the paralyzed limbs when those parts were manipulated; a condition which approaches in a way the dysesthesia of thalamic disease.

In Case 4, which was that of a soldier who suffered a head wound, the return of function of rectum, bladder and sexual apparatus first appeared with return of movement of the hips, and a short time afterward was normal.

In a person clear mentally, paralysis of the bladder, bowel and sexual function probably never occurs from a unilateral cortical lesion, although some cases of brain tumor limited to one side do show this phenomenon; but in most of these cases there is considerable mental derangement.

Concerning the functions of the bowels, bladder and sexual functions, from the data I have presented, it seems logical to assume that a cerebral condition must be bilateral to produce paralysis of these functions, the centers for which are in close proximity to the cortical areas which control the hips.

DISCUSSION

Dr. Charles K. Mills, Philadelphia: Dr. Wilson's paper is one of the few clinicopathologic communications bearing on the location of the cortical centers of the bladder, bowels and sexual functions. I think very few cases of bladder incontinence from cortical disease have been reported. Sometimes we make the mistake of attributing to mental hebetude incontinence of bladder and bowels which are really due to involvement of centers concerned with these functions. This paracentral region is far removed from the thalamus, and, apparently, in Dr. Wilson's cases no local conditions were present to account for the pain in the limbs. His paper suggested the question whether or not the cortical centers for pain in the limbs may not have been involved in these cases. One of the theories held by Horsley, Schaeffer and others is that the centers for pain are, in part at least, located in the limbic lobe near the paracentral region.

THE PSYCHIC MANIFESTATIONS IN PARALYSIS AGITANS

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AND
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While paralysis agitans is not a common affection, its frequency being put by the majority of observers at about 1 per cent. of all nervous diseases, it has been recognized as a distinct clinical entity since 1817, when its neurologic syndromes were first described by Parkinson, a well developed case of "shaking palsy" presents no difficulty in the matter of diagnosis.

It would seem, however, that the psychic manifestations of this disease have to a great extent been overshadowed by the neurologic symptoms; most writers dismiss the mental changes with the statement that there may be present in this disease some irritability and in cases occuring late in life some defects of memory, while still other observers assert that as a rule the mind remains entirely clear.

Unquestionably, there are many cases of paralysis agitans that are uncomplicated by well marked mental symptoms; but, on the other hand, there are patients suffering from this disease who present definite evidences of a psychosis and when this occurs the mental syndromes are as characteristic and constant as are "the mask-like expression and pill rolling fingers." Not only is this true, but mental symptoms may be the first to make their appearance and may precede by months or years the classical neurologic signs.

From the psychiatric standpoint, the etiologic factors in themselves are such as to suggest the probability of abnormal psychic functioning as part of the disease syndromes. Heredity would seem to play a part as a predisposing cause, as a neuropathic ancestry is frequently encountered and in some instances the disease appears in several generations or in collateral family branches; the direct exciting causes are as a rule psychogenic in nature and the first manifestations of the disease frequently appear after undue or prolonged anxiety, fear, or grief; and while in some cases traumatism has apparently incited the condition, in these cases the element of mental shock cannot be excluded.

The onset of the mental symptoms in this psychosis is as a rule gradual with prodromata referable especially to the emotional reactions.

The patient shows evidences of nervousness, is easily irritated, worries over trifles occurring in connection with his family and his work; he becomes introspective and suspicious, may complain of vague, ill defined pains and often is unable to sleep. These early manifestations may continue for weeks or months and slowly merge into more well defined manifestations of a true psychosis; this psychosis, if it occurs before the development of well marked neurologic signs, frequently leads to the diagnosis of manic-depressive insanity, involution melancholia, or senile psychosis, which it may closely simulate.

In the fully unfolded psychosis, all psychic factors are not equally affected, the most marked involvement being found in the fields of perception, judgment and affectivity.

Disorders of attention, if they occur at all, are slight. There may be marked inadequancy of involuntary attention as a result of introspection and absorption by delusions, but this does not reach a true hyperprosexia and the patient is generally found in touch with most of his environmental happenings. Voluntary attention can be easily gained, the thought processes are capable of adequate direction and concentration as a rule is good.

The stream of mental activity, if at all altered, shows diminished productivity. There may be little spontaneous conversation, but answers to questions are coherent and relevant, and while the utterances may be slow, investigation will show that there is no well marked mental retardation; the slowness of speech is a forerunner of the monotonous scanning type that later is to become so definitely established.

The mental trend is always depressive and the thought content is dominated by false percepts and concepts. Hallucinations are frequent; they are generally limited to the organic sensations and tactile sense, and are generally the result of sensory and vasomotor disturbances. The patient will complain of strangulation and blocking of the intestines, disappearance of the stomach, electricity continually passing through his arms and legs; on these false percepts are developed well defined delusions of a somatic, self accusatory or paranoid nature. The patient will refuse food and interpret all efforts to compel him to take sufficient nourishment as attempts to put him out of the way; members of his immediate family or unknown persons are responsible for the electric currents that pass through him, or he will think he has committed an unpardonable sin and is responsible for the misfortunes of others about him.

The emotional state is in keeping with the mental content. These patients are always emotionally depressed, at times markedly agitated, give evidence of great psychic pain and not infrequently this condition leads to attempts at suicide or homicide.

Consciousness is as a rule clear, orientation is correct and, save in the late stages of the disease, there is but little mental confusion.

Memory is well retained as a rule; though mental inertia is present, fixation, conservation and reproduction are generally good; the exception to this is in those patients in whom the disease occurs quite late in life; then there may be amnesia, the result mainly of general senile brain changes.

The following cases studied at the Danville State Hospital, Danville, Pa., serve to illustrate the mental manifestations of paralysis agitans as set forth above.

REPORT OF CASES

Case 1.—D. S., a woman, aged 40, Russian by birth, married, gave a history of nervousness and hysteria in her ancestry; also, one brother died with general paralysis in the Danville Hospital. The patient had always been of a neurotic temperament. The first mental symptoms were manifested four years before admission, when, following a mild attack of influenza, she became emotionally depressed and hypochondriacal. Two years later, while pregnant, she developed a neuritis in both upper and lower extremities which disappeared after the delivery of the child. A few months later she suffered from pleurisy and after several months of illness, while emotionally depressed, she attempted suicide by shooting. Shortly after this her husband developed a malignant disease; the patient at once became markedly agitated, self accusatory, believed her husband's condition to be an infliction of divine wrath in punishment for her sins, and while in this condition she again attempted suicide by drinking "Lysol." At this time, four years after the development of mental symptoms, neurologic signs first appeared, and the case was diagnosed by Dr. F. X. Dercum as one of paralysis agitans. She has been under treatment in the Danville State Hospital for six months and at the present time is emotionally depressed, self accusatory, and actively suicidal; but, in spite of the fact that the disease has progressed to the point when she is confined to bed, there is no mental retardation, clouding of consciousness or memory change.

Case 2.—R. R., a married man, aged 56, a coal merchant, with a family history that was negative so far as known, had been of good natural intelligence, temperate habits, and economically efficient, and had always enjoyed good physical health. He was apparently free from mental disorder until six years before admission, when, following business reverses, he became nervous, emotionally depressed and hypochondriacal. This condition continued for six months when a serious injury to his favorite son completely upset him mentally; delusions of a self accusatory, paranoid and somatic character developed and while in this condition he attempted suicide by hanging. He was admitted to the Danville State Hospital where he remained under treatment for three months and thirteen days. During this time, he complained of pain and numbness in the right arm and leg, but no objective neurologic signs were present. Emotional depression was marked and delusions of a paranoid and somatic character were very prominent. He refused food because his stomach and intestines were blocked and when urged to eat, he accused the physicians and nurses of feeding him for the purpose of causing his abdomen to burst. Gradually he improved and his psychosis was diagnosed as manic-depressive, depressed phase. He returned to his home and resumed work; while he continued to show periods of mild depression, undue worry over business trifles

and at times decided irritability, he adapted himself fairly well to his environment for nearly five years, when, gradually, tremors of the right hand developed, the face became expressionless and speech showed marked defects. He was examined by Dr. F. X. Dercum and the condition was diagnosed as paralysis agitans. Gradually, his old delusional manifestations reappeared and at the present time he is again in the Danville State Hospital showing, in addition to the neurologic signs, extreme mental depression with agitation and marked psychic pain, delusions similar to those already noted; consciousness remains clear and there is entire absence of intellectual deterioration.

Case 3.—E. W., a widow, aged 63, concerning whose ancestry no reliable history was obtained. She was admitted to the hospital with a history of periods of emotional depression of rather short duration extending over several years. These attacks were mild in character and did not prevent her from caring for her family and, although she was widowed and without financial means, she educated her five children. Three months prior to admission, she became greatly worried over the drafting of her son into the army and gradually developed delusions of persecution against her neighbors, who, she believed, were plotting to do her bodily harm; in consequence she threatened to kill her supposed enemies. On admission to the hospital, she showed marked physical prostration with myocardial weakness, but an entire absence of neurologic signs or symptoms.

Mentally, there were present: great emotional depression, agitation and psychic pain, with pathologic irritability, auditory hallucinations and well fixed, systematized delusions of a paranoid nature—nurses and physicians were plotting against her life; poison was placed in her food. For several months it was necessary to resort to tube feeding to sustain life. Memory showed some slight impairment, especially a lack of fixation and conservation of recent happenings. Fifteen months later she complained of numbness and tingling in the arms and legs; she attributed them to electrical currents which her persecutors were passing through her. Six months later, or twenty-one months after admission to the hospital, the first well marked neurologic signs developed and the condition which had been diagnosed as a senile psychosis had become a typical case of paralysis agitans.

Case 4.—L. H., a widowed woman, aged 71, concerning whom no reliable data as to ancestry or early life were available, gradually developed a mental disorder, extending over a period of two years, which was characterized by emotional depression, delusions of persecution directed against her neighbors, threats of homicide and gradual loss of memory. On admission to the hospital, she presented the mental picture of a senile psychosis and her case was so diagnosed. She continued in a depressed emotional condition and exhibited well defined delusions of a persecutory nature for a period of two years; then, gradually, neurologic signs of paralysis agitans appeared and rapidly progressed. The patient became confined to bed and died as the result of a terminal bronchopneumonia, four years and seven months after admission.

Case 5.—G. S., a liveryman, aged 62, whose father was insane at the time of death and one of whose brothers died in this hospital, had been normal mentally and physically until seven years before admission when he first complained of a twitching of the right great toe. The twitching gradually extended to the leg and arm of the same side and later involved the left side. As this condition progressed, emotional depression became marked; delusions of a paranoid nature developed and the patient became homicidal. He believed his food was poisoned and refused to eat. On admission to the hospital, he was

in a critical physical condition, with evidences of starvation together with the neurologic signs of Parkinson's disease. He failed to respond to treatment, rapidly passed into a condition of coma and died five days after admission. Necropsy revealed no definite pathology other than chronic parenchymatous myocarditis, chronic parenchymatous nephritis and bilateral hypostatic congestion of the lungs.

CONCLUSIONS

1. From the cases cited, it may be seen that mental manifestations are not uncommon in paralysis agitans and that they are frequently overshadowed by the neurologic symptoms.

2. The mental symptoms in paralysis agitans often precede the neurologic signs and lead to erroneous diagnoses.

3. Mental manifestations in paralysis agitans are characteristic, and consist of emotional depression; agitation and psychic pain; hallucinations, generally referable to the organic sensations and the sense of touch; delusions of a somatic, self accusatory or paranoid nature with resultant attempts at homicide or suicide; and varying defects of memory with little mental confusion.

News and Comment

ASSOCIATION FOR RESEARCH IN NERVOUS AND MENTAL DISEASES

Fourth Annual Meeting, December 27 and 28, 1923

HOTEL COMMODORE, NEW YORK

PROGRAM

1. Introductory Address on the general subject of heredity.
DR. WALTER TIMME.

2. The characteristic of heredity and inherited constitutional predisposition.
DR. CHARLES L. DANA.

3. The biologic mechanism of heredity. Contributor to be announced.

4. The parts of the central nervous system which tend to exhibit morbid recessive or dominant characters. DR. SMITH ELY JELLIFFE.

5. Is there fundamental similarity in the pathological processes which occur in the different parts of the central nervous system in the various hereditary nervous and mental diseases? DR. LEON H. CORNWALL.

6. Is there fundamental similarity in the pathological processes which occur in the extraneural systems of the body in the various hereditary nervous and mental diseases? DR. NOLAN D. C. LEWIS.

7. The diseases and accidents occurring during pregnancy, labor and early childhood which if unrecognized would lead one to believe that the resulting condition was a hereditary disease. DR. CHARLES HENDEE SMITH.

8. Biochemistry of hereditary diseases. Contributor to be announced.

9. Experimental production of degeneracy in its bearing on hereditary abnormalities of the nervous system. DR. CHARLES R. STOCKARD.

10. The hereditary factors responsible for the development of optic atrophy and retinitis pigmentosa. DR. WILLIAM H. WILMER.

11. The hereditary factors operative in the development of amaurotic family idiocy. DR. BERNARD SACHS.

12. The hereditary factors in the development of the psychoses.
DR. ALBERT M. BARRETT.

13. A discussion of the factors entering generally into the development of hereditary diseases such as consanguinity, race, endocrinopathies, toxins, etc.
DR. LEWELLYS F. BARKER.

14. A paper outlining the evidences of hereditary disturbances throughout the animal and plant kingdoms. DR. HENRY F. OSBORN.

15. Pathology of hereditary nervous and mental disease.
DR. SAMUEL T. ORTON.

16. Heredity in literature. DR. JOSEPH COLLINS.

DIETS AND FASTING IN EPILEPSY

The predominatingly toxic nature of the causes of symptomatic and experimental epilepsy leads naturally to the suspicion that some toxic substance is responsible for the fits of idiopathic or essential epilepsy. The intestine is one possible site for the origin of such toxins; it is conceivable that they could arise from faults in digestion, from fermentation produced by bacterial growth, or in other ways. The hypothesis that they arise by fermentation has led to measures directed toward changing the flora of the intestinal tract, sometimes by heroic means, but so far without lasting therapeutic success. Efforts to control epilepsy by dietary regulation have for years been attempted, and heretofore have been largely empiric. It is a common custom to limit the consumption of meat by epileptics on the assumption that excessive protein and particularly excess of purins favor the occurrence of "fits." Within the last two years, prolonged abstinence from food of all kinds has been somewhat extensively exploited as a cure for epilepsy, and has been the subject of special study. Carefully controlled investigations of the effects of variations in the quantities and composition of foods in epilepsy are therefore especially welcome.

Weeks, Renner, Allen and Wishart,[1] working with patients under complete control at the State Village for Epileptics, Stillman, N. J., have studied the effects of complete fasting and also of a nonnutritive bulk diet continued for periods of three weeks. In many cases the fits decreased in number, in some they ceased entirely, in others there was no change in frequency. In general, after the termination of the fast, the attacks recurred as frequently as before. Similar results from starvation have been reported by Goldbloom.[2] Lennox[3] observed decrease in the number of fits under similar conditions, and reports that studies of the nonprotein nitrogen in the blood, which had been uniformly within normal limits before the fast, revealed a marked increase, especially of the uric acid fraction. The conclusion, therefore, seemed justified that fits were not caused in these cases by a large excess of uric acid or other nonprotein nitrogenous compounds in the blood.

Weeks and his co-workers also studied small groups of patients who were maintained for several weeks on high protein (up to 260 gm., with 32 gm. of fat), high calory (over 8,000), high carbohydrate (from 500 to 800 gm.) and high fat (from 260 to 440 gm.) diets. They concluded that none of these diets had a demonstrable relation to the frequency of the fits. These results are somewhat disconcertingly at variance with generally prevailing opinion. They conflict with the observations reported by Cuneo, to which reference was made in an earlier editorial,[4] that the incidence of fits was much greater with a high carbohydrate than with a high protein diet.

It seems, therefore, that at present there is no justification for exploiting any particular dietary regimen for general application in the management of epilepsy. Common sense avoidance of materials that give rise to gastric or intestinal indigestion, with due regard for individual idiosyncrasies, must con-

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1. Weeks, D. F.; Renner, D. S.; Allen, F. M., and Wishart, Mary B.: Observations on Fastin and Diets in the Treatment of Epilepsy, J. Metabol. Res. **3**:317 (Feb.) 1923.

2. Goldbloom, Alton: The Starvation Treatment of Epilepsy, Canadian M. A. J. **12**:539 (Aug.) 1922.

3. Lennox, W. G.: The Chemistry of the Blood in Epilepsy, J. A. M. A. **81**:1138 (Sept. 29) 1923.

4. Research in Epilepsy, editorial, J. A. M. A. **80**:1939 (June 30) 1923.

tinue to guide the selection of the appropriate diet, in epilepsy as well as in other diseases. Particularly should it be emphasized that, while it is true that the number of fits often diminishes during a period of starvation, there is no justification for claims that this measure is in any sense a cure for epilepsy; the remarkable changes in the blood chemistry, as well as common sense, lead to the suspicion that it may be a cause of potential, if not immediate, actual damage.—*Edit. Jour. A. M. A., Oct. 6, 1923.*

SECOND ANNUAL CONVENTION OF THE CENTRAL NEUROPSYCHIATRIC ASSOCIATION, ST. LOUIS, OCTOBER 20, 1923

The morning session, at Barnes Hospital, began with a neurosurgical clinic by Dr. Ernest Sachs and assistants.

Dr. Greenfield Sluder, rhinologist, read a paper on Sphenopalatine Neuralgia and Lower Half Headaches. Dr. Sidney Schwab and assistants (Dr. A. B. Jones, Dr. A. D. Carr, Dr. G. B. Smith) presented eight cases in a general neurologic clinic, consisting of: (1) glioma, frontal lobe; (2) tic or cortical fits following mumps in a man aged 72; (3) suprasellar tumor; (4) left cerebellar lesion invading the meninges; (5) paretic seizures with respiratory failure; (6) muscular atrophy with subjective sensory symptoms; (7) mental depression with a series of conscious conflicts and an inferiority complex, and (8) case showing the submergence of psychic symptoms by minor complaints and minor findings.

A clinic of neurosyphilis in children was conducted by Dr. P. C. Jeans.

The afternoon session, in St. Johns Hospital, was opened with an endocrine clinic by Dr. Wm. Engelbach, followed by an illustrated discussion of pituitary disease.

Psychiatric papers were presented as follows:

Mental Conditions Associated with Deafness. Dr. L. B. Alford.

The Psychiatric Factor in Vocational Incapacity. Dr. F. M. Barnes.

The Development of a Mental Hygiene Program in St. Louis. Dr. M. A. Bliss.

Report of a Family of Four Generations Presenting Sixteen Cases of the Hypertrophic Neuritic Type of Progressive Muscular Atrophy. Dr. M. L. Perry, (Topeka, Kans.).

The annual dinner was held at the University Club. Dr. Arthur S. Hamilton, of Minneapolis, made the presidential address dealing with available facilities for special training in neuropsychiatry, and Dr. Hugh T. Patrick, of Chicago, gave a survey of European neurology as observed in a recent trip abroad.

The officers elected for the succeeding year are:

President, Dr. George A. Moleen, Denver; Vice President, Dr. Herman M. Adler, Chicago; Secretary-Treasurer, Dr. Karl A. Menninger, Topeka; Counsellor, Dr. Arthur S. Hamilton, Minneapolis.

Twenty-six neurologists and psychiatrists of the central and western parts of the United States joined the association. Number of members present, 40; total attendance, 85.

The Central Neuropsychiatric Association was formed to afford better acquaintanceship among the neuropsychiatrists of the central and western states and for mutual improvement. Annual meetings are held at which the men in the convention city furnish the program. This is expected to be principally clinical.

Abstracts from Current Literature

MORPHOGENESIS OF THE NERVOUS CENTERS IN CHIROPTERA. PART I. THE FIRST PHASES OF DEVELOPMENT OF THE NERVOUS CENTERS IN RHINOLOPHUS HIPPOSIDEROS (BECHSTEIN). E. Luna, Folia neurobiol. **12**, 1922.

The development of the central nervous system in *Chiroptera* has been little studied. Many problems related not only to the morphogenesis of the organs which are differentiated from the vesicles of the neural tube but also to the general morphogenesis of the neural tube itself, remain still unsolved. The author, being in possession of a rich embryonic material, has undertaken comparative embryologic researches in several species of *Chiroptera*, namely *Rhinolophus hipposideros* (Bechstein), *Vespertilio murinus* (Schreb), *Vesperugo Kuhlii* and *Miniopterus*. Part 1, dealing with the first phases of development of nervous centers in *Rhinolophus*, has been published in full in Ricerche di Morfologia, Vol. 2, Rome. It follows the evolution of the nervous centers from the epoch of the closure of the neural tube up to the rudiment of the cerebral hemispheres.

The methods of Spuler, Zenker and others, staining with hematoxylin, hemalum, congo red, etc., were employed. The embryos were cut serially in frontal, transverse and horizontal planes. The method of plastic reconstruction with the Born-Peter apparatus was extensively used. Very good results were also obtained with the demolition method of Chiarugi. There are fifty-nine good figures in the text and a complete bibliography of the subject closes the article. A minute description of all embryonic stages, from Stage 1 to Stage 7, with measurements and all the transformations, precedes the general summary.

FLEXURES OF THE ENCEPHALIC AXIS

In the embryonic brain of *Rhinolophus* three flexures are early differentiated; the cephalic or apical (Scheitelkrümmung), the nuchal (Nackenkrümmung), and the pontile flexure (Brückenkrümmung). The bending of the neural tube in its anterior segment cannot be interpreted as the consequence of the growth of the pontile flexure, contrary to the opinion of Tandler who attributed it to the volumetric increase of the prosencephalon. The nuchal flexure appears in emryos of Stage 1 as a slight curve dividing the rhombencephalon from the spinal cord. In the embryos of Stage 3 the axis of the rhombencephalon forms with that of the spinal cord an obtuse angle which is gradually reduced approaching the size of a right angle in embryos of Stages 6 and 7. The pontile flexure appears relatively late in embryos of Stage 3. In later stages it becomes very prominent, the base of the rhombencephalon coming almost in contact with the infundibulum. This condition is different from what is found in birds, where the flexure appears in much later stages and does not reach a great development.

DIVISION OF THE ENCEPHALON AND LIMITS OF THE SINGLE CEREBRAL VESICLES

There is no doubt as to the existence in *Rhinolophus* of the primary differentiation of the three vesicles; prosencephalon, mesencephalon and rhombencephalon. There is difference of opinion, though, regarding the successive sub-

divisions of the neural tube and the delimitation of the various vesicles resulting from it. The author has failed to recognize in the first phases of development of *Rhinolophus* an isthmus of the rhombencephalon between mesencephalon and metencephalon, as described by His.

Myelencephalon and metencephalon originally are not distinct from one another as are the other segments of the neural tube; they never assume the shape of two seperate vesicles. The anterior limit of the mesencephalic base corresponds to the tuberculum posterius, and perhaps more exactly, to the small pit which lies before it. As is known, there is no consensus of opinion either on the limits between diencephalon and telencephalon, or as to the allotment of that region of the neural tube from which the optic vesicles are formed, to either one of the vesicles originating from the primary anterior cerebral vesicles. The author has found that from the telencephalon three zones are early differentiated; telencephalon, diencephalon and opththalmencephalon. However, in the progress of development, the limits of the ophthalmencephalon become steadily less evident until they disappear entirely, the opththalmencephalon becoming a portion of the diencephalon. Contrary to the opinion of Tandler and Kantor it cannot be admitted, at least in *Rhinolophus*, that the infundibular portion is a segment primarily developed from the prosencephalon and therefore differentiated from the rest of the diencephalon; in no stage of development does a demarcation between the two portions exist.

THE SPINAL CORD

In Stage 1, the spinal cord has the shape of a tube whose anteroposterior diameter is larger than the transverse. The caliber is even throughout the whole length of the cord, except for a diminution at its caudal end where it assumes the cylindric form. In this stage the caudal end presents a large dorsal fissure which is the posterior neuropore.

The lateral walls are thick and connected by means of two thin ependymal laminae, a ventral or basal plate and a dorsal or roof plate, the latter thinner than the former. The central fissure, namely the cavity of the spinal cord, which precedes the definitive central canal, appears, in a transverse section, elongated in its sagittal diameter, narrower in its middle portion, with two enlargements on the sides, the dorsal being the larger. Proceeding caudad the fissure has the form of an ovoid with the posterior pole bigger than the anterior. The basal plate is constituted of two or three layers of elliptic nuclei, whose greater axes have a sagittal direction. In the roof plate the nuclei are round and disposed in a single layer. The lateral walls are constituted of indifferent nuclei (indifferent cells of Schäfer) disposed in six or eight planes, which constitute the internal plate (Innenplatte of His) or layer of the nuclei (Kernschicht). Outside these there is a narrow marginal layer, which represents the rudiment of the white substance, whose initial formation is therefore quite precocious. The germinative cells are disposed near the central fissure. The marginal layer, absent in both the basal and tectal plates, appears in successive stages. In the caudal section of the cord the indifferent cells are more scarce and the marginal layer is almost entirely absent.

In Stage 2, slight horizontal depressions, segmentally disposed, and separated by regular projections of the neural wall, begin to appear on the external surface of the cord. This is the first outline of the neuromeres. A ventricular neuromery corresponds to the superficial neuromery. Almost unchanged

remains the form of the central fissure, except that its transverse diameter tends to become a little larger. The posterior neuropore is either closing or almost entirely closed. The indifferent cells, and the marginal layer are increased.

In Stage 3 the protoplasmatic fibrils appear on the free surface of the ependymal cells, as contractile cilia, especially in the cells of the basal plate.

The neuromery of the spinal cord is preserved in Stage 4, but in Stage 5 it disappears almost entirely. In this last stage a wide longitudinal groove appears in the wall of the central fissure, namely the so-called sulcus limitans, and in the lateral walls an alar plate and a fundmental plate. The author was unable to distinguish a portio intermedia or Schaltstück of His, because neither in this stage nor in the following stages was a posterior lateral sulcus recognizable. The structural differentiation of the lateral walls becomes more evident is Stage 5 and even more so in Stage 6, in which a modification of the shape of the cord takes place. This, in transverse sections, appears as an irregular quadrangle having two lateral faces, one dorsal and one ventral. In the point where the lateral face passes to the dorsal, superficial projections appear on account of the development of the dorsal columns and of the tractus ovalis, which represents the rudiment of the posterior funiculus. Another white formation appears well pronounced in this stage, the ventral commissure, which is formed by the arcuate fibers (arcuate formation of His) running among the cells of the anterior horn and intersecting in the middle line with the ependymal fibers of the basal plate. The evolution of the spinal cord proceeds from the cranial toward the caudal region. The development of the cervical and lumbar enlargements appears very early in the embryo of 5 mm. The closure of the anterior neuropore precedes shortly that of the posterior neuropore, the one taking place in Stage 1, the other in Stage 2.

In connection with the spinal neuromery the author cites Sterzi (whose results in fishes are in accord with his own), Froriep, Widersheim, Dorello, Lucy and Herrick, the latter attributing the segmentation of the spinal cord to mechanical effects produced by the formation of the rudiments of the nerves. He remarks that in *Rhinolophus* the neuromeres appear before the ganglions manifest themselves as definite formations, capable of determining pressure on the surrounding organs, and that their correspondence with the somites is very clear. He thinks it is very likely that in the spinal cord a true metamery in the morphologic sense does not exist, that it is due to pressure exerted by the primitive segments.

RHOMBENCEPHALON

The division of the rhombencephalon into metencephalon and myelencephalon is only possible because the dorsal wall at the level of the metencephalic segment appears thicker than in the myelencephalon where it will form the myelencephalic tela chorioidea.

In Stage 1 the myelencephalon has the form of a very narrow tube, slightly flattened on the sides. The walls of the myelencephalic ventricle are smooth, except for the region which will constitute the tela chorioidea where the dorsal plate is thinner and made up by very flat cells. In this stage the myelencephalon has the same aspect and structure as the spinal cord. In Stage 2, embryo of 2 mm., the myelencephalon undergoes transverse expansion and becomes the widest segment of the neural tube. The myelencephalic cavity shows a different appearance in the different transverse sections. These differences are in relation with the progressive broadening of the roof plate

(future tela chorioidea), which near the metencephalon is much wider than elsewhere. In Stages 3, 4 and 5, the expansion of the transverse diameter of the myelencephalic ventricle gradually progresses, until it reaches its highest degree at Stage 6, in which the intermediate segment, the region of the calamus scriptorius and the region of the maximal width of the rhombic cavity (losange) can be distinguished. The first outline of rhombomery appears in Stage 2; therefore it is contemporaneous with the so-called neuromery of the spinal cord. The rhombomeres in *Rhinolophus* are six. The first, however, belongs to the metencephalon, which in this stage begins to be differentiated from the myelencephalon, by the thickening of the roof (cerebellar neuromere—Kleinhirnneuromer). In the following stages the rhombomery gradually disappears, the external one first. In Stage 7 (embryo of from 5.5 to 6 mm.), while the external surface is regular, the ventricular surface shows, in the depressio transversa rhombi, three transverse grooves which limit two small transverse ridges. Besides, there are also longitudinal grooves, two for each side, which bound three ridges corresponding to the eminentiae longitudinales rhombencephali described by Kupffer in *Bdellostoma Scyllium* and other craniota. The grooves are medial and lateral; the latter is very likely the sulcus limitans. As to the probable significance of the rhombomeres the author has postponed his final opinion until after the study of other species. A discussion of the modifications which the dorsal margin of the alar plates of the myelencephalon, or rhomboidal lip (Rautenlippe of His) undergo follows the findings of Herrick, Blacke, Charnock Bradley, Dexter, Groenberg and Vernoni, who studied it in different species. The author found in the cranial segment of the myelencephalon and also in the metencephalon an eversion of the rhomboidal lip with the formation of an internal and external groove. Histogenetic researches have shown that in embryos from 1.5 to 2 mm., the structure of the myelencephalic walls is almost similar to that of the spinal cord. It is in embryos of 2.5 mm. that the differentiation into three layers—germinal, mantle, and marginal—begins. In Stage 5, embryo of 4 mm., the mantle layer is thicker and the elements of the nucleus of the twelfth nerves are particularly evident. The marginal layer is still thin, except in the fundamental plate. The basal plate is constituted by several layers of spongioblastic nuclei with germinal cells. The spongioblasts form the septum medullae with their divergent processes. The tectal plate is reduced to a layer of flat cells, outside which is the mesenchyme, which forms the rudiment of the meninges. It is in the later stages that the septum medullae is crossed by nerve fibers going from one side to the other with the formation of a real raphe. In these stages neuroblasts and glioblasts of the mantle layer of the alar zone migrate toward the ventral region. The same phenomenon is observed where the soldering of the external labial sulcus takes place, neuroblasts and glioblasts moving laterally to a segment of the marginal layer, which represents perhaps the rudiments of the tractus solitarius. The descending radix of the trigeminal is clearly seen in the embryo 3.5 mm. long.

The metencephalon appears in the embryo of 2 mm. as a differentiated segment of the neural tube in the form of a short vesicle. The distinction between metencephalon and myelencephalon appears only when the sketch of the plica chorioidea begins, namely in embryos from 5.5 to 6 mm. long. A cerebellar portio and a pontile portio can be distinguished very early. The lateral walls of the metencephalon are, in fact, contributed by a dorsal segment (alar plate or cerebellar plate) and by a ventral segment (pontile plate) separated by the

sulcus limitans. The two cerebellar plates are united by means of the tectal plate, the pontile plates are united by means of the basal plate. The two pontile plates from a sagittal disposition assume a gradually more horizontal direction; they also become thicker with the appearance of a mesial longitudinal furrow (future sulcus basilaris). The histogenetic differentiation of the pons follows the development of the other portions of the neural tube. The two pontile plates, first formed by the layer of nuclei and by the thin marginal layer, present three distinct layers, germinal, mantle, and marginal, in the embryo 5.5 mm. long.

The cerebellar plates also have a sagittal direction in the first stages. In the successive stages they assume a transverse disposition. In embryos from 5.5 to 6 mm. long the cerebellum appears as a transverse plate behind the mesencephalon, from which it is separated by means of the deep rhombomesencephalic fossa. The cerebellar laminae undergo a gradual increase in thickness, protruding into the underlying ventricle. The roof plate, at first thin, gradually reaches the thickness of the alar plates; therefore the rudiment of the cerebellum becomes single and medial. The cerebellar plates are also first originated as in the other portions of the neural tubes by a layer of nuclei and by a thin marginal layer. The differentiation in the three layers begins in the embryo of 4 mm., except for the velum medullare anterius which contains only the germinal layer. This structure is preserved in embryos of 5.5 mm., in which the rudiment of the cerebellar body presents a very thick mantle layer formed by an internal nuclear layer, an internuclear layer and an external nuclear layer, the latter corresponding to the layer of Obersteiner. This, contrary to the opinion of Sterzi, originates from the germinal layer; therefore from the morphologic point of view the lip of the cerebellar margin must be considered as the equivalent of the rhomboidal lip.

DEVELOPMENT OF THE RHOMBENCEPHALIC NERVES

The radicular fibers of the trigeminal appear very early in embryos of 2.5 mm. They are seen to emerge from the ventrolateral surface of the rhombencephalon, immediately behind the rhombomesencephalic sulcus. More caudad in a ventral plane is situated the radix of the sixth nerve, which together with the seventh and the eighth nerves, originates from the rhombencephalic segment that may be considered as a myelencephalic portion. Still more caudad are the roots of the ninth and tenth nerves, and at the point of transition of the myelencephalon into the spinal cord emerge the roots of the twelfth nerve situated in a ventral plane. The eleventh nerve is not evident in this stage. In the embryos 5.5 mm. long the apparent origin of the fifth nerve is situated at the limit between metencephalon and the point of maximal width of the rhombencephalon. Slightly caudad, but in a ventral plane, appear the roots of the sixth nerve, which is now decidedly a myelencephalic nerve. The same may be said for the seventh and eighth nerves. The roots of the ninth, tenth and eleventh nerves come after. The twelfth nerve emerges at the same level as the eleventh, but in a more ventral plane. The author thinks that in *Rhinolophus*, as was found by Kappers in other superior craniota, a migration to the metencephalon of the same nuclei takes place, which in the embryonic stages are myelencephalic. Sterzi showed that in man also at the end of the first month the trigeminal emerges at the point of limit between myelencephalon and metencephalon.

MESENCEPHALON

In Stage 1, the mesencephalon has about the same general outline and structure as the other parts of the neural tube. In the embryo of 2 mm. (Stage 2) two mesomeres appear; their formation is contemporaneous with neuromeres of the rhombencephalon. In the same embryo appears also the sulcus limitans which, in this stage, is an exclusive formation of the mesencephalon and of the cranial portion of the rhombencephalon. In embryos 2.5 mm. long (Stage 3) the roots of the third nerves make their first appearance; the mesencephalic base is somewhat thickened by the presence of the tori tegmentales, whose rudiments, however, began to form in the preceding stage. In embryos 4 mm. long (Stage 4) a mantle layer begins to differentiate, with the appearance near the medial line of neuroblasts which constitute the nucleus of the oculomotor. In stage 5 the groove and the projection which divide the two mesomeres disappear. It is in embryos from 5.5 to 6 mm. long (Stage 7) that the mesencephalon begins to present important changes owing to the development of the vault, which expands into the two lateral vesicles or optic lobes. In this stage the radicular fibers of the trochlear are seen emerging from the rhombomesencephalic sulcus behind the mesencephalic roof. A longitudinal spur like the one described by His in man is found along the middle line of the roof. The vault is constituted by the germinal layer and by a very thin marginal layer; in the lower portions of the optic lobes, however, a conspicuous mantle layer and a large marginal layer are found. The development of the mesencephalic base proceeds simultaneously with that of the vault; its behavior varies though from the cranial to the caudal segments. The caudal segment is formed by thick germinal and marginal layers; scarce neuroblasts between the two represent the mantle rudiment. More cephalad the fusion of the mesencephalic base with the optic lobes takes place. A small opening in embryos of 5.5 mm. indicates that the fusion is not complete. The nucleus of the oculomotor is not well differentiated.

PROSENCEPHALON AND DIENCEPHALON

In embryos 1.5 mm. long, the diencephalon appears as a vesicle flattened on the sides and elongated in the anteroposterior direction; its posterior pointed end corresponds to the future region of the infundibulum, whose differentiation begins in the embryos 2 mm. long. The diencephalic vesicle appears somewhat dilated; it presents two lateral walls, a base and a roof. There is no indication of the sulcus limitans, but the posterior commissure begins to form in the vault. This commissure develops in part in the diencephalon and in part in the mesencephalon, a condition similar to that found by D'Erchia in fishes, guinea-pigs and in man. In Stages 3, 4, and 5 the infundibular region assumes gradually greater proportions, but only in Stage 6 does the encephalic vesicle begin to show special peculiarities in form, owing to the first outline of the optic thalamus appearing on its external lateral walls. The thalamus seen from the inside has the appearance of a hoop elongated in its anteroposterior diameter. It is bounded by two furrows, a rostral one which is the striothalamic sulcus and a dorsal one which is the sulcus limitans, or Monro's sulcus, dividing the thalamus from the hypothalamus. It terminates in the optic recess. The author confirms the interpretation of Vernoni, namely, that the sulcus opticus or the radicular sulcus of the optic vesicle is divided into two other sulci, the sulcus Monroi and the sulcus striothalamicus. The diencephalic roof presents a thin medial band which is the vault of the third

ventricle, and two lateral zones which are the epithalami. In Stage 7 a slight depression of the internal or ventricular layer of the vault indicates the first sketch of the epiphysis. This mode of development of the pineal body was also found by Chiarugi in the guinea-pig and by Vernoni in *Muletia novemcincta*. In Stage 7, also, the two thalami coalesce in the middle commissure (commissura mollis). Such commissure appears therefore quite early in *Rhinolophus*. On account of a gradual diminution of the base of the optic vesicle, the ophthalmencephalon assumes the appearance of an appendix of the diencephalon. The regio optica seen from the inside is represented by the optic or preoptic recess, which laterally ends in the optic peduncles. The postoptic plate and the postoptic recess do not belong to the regio optica but to the region of the infundibulum. The chiasmatic protuberance appears early as a slight ventricular projection of the neural wall. It is the primitive chiasmatic protuberance and represents the ventricular expression of the postoptic sulcus. Later, for the development of the fibers of the optic tract, it will become the definitive chiasmatic protuberance. The histogenic differentiation of the diencephalon begins rather late in *Rhinolophus* in comparison with man. The three layers are well distinguished in the embryos of 5.5 mm. but their development is not the same in the different parts of the lateral walls. In the embryos from 5.5 to 6 mm. a projection of the ventricular wall appears ventrad to the mammillary recess. It is a formation found in teleosts, reptiles and birds, and corresponds to the tuberculum mammillare of Kupffer or tuberculum postinferius of B. Haller.

TELENCEPHALON

In some embryos of Stage 1, a telodiencephalic sulcus appears, which continues caudad with the preoptic sulcus. The telodiencephalic prominence and the preoptic prominence are the ventricular expression of these two sulci. In a sagittal section of the neural tube the telodiencephalic prominence appears as a thickening of the wall. It is the first outline of the velum transversum, whose existence in mammals is denied by some authors. It is very evident in the successive stages of *Rhinolophus*. In Stage 2, two sulci tegmenti appear which outline the evagination of two lateral vesicles. In Stage 3, the telencephalon begins to differentiate as a cerebral vesicle; its division in singles and pairs becomes more evident. In Stage 5 appears a neuroporic recess; the lateral vesicles expand on the sides of the nonpaired telencephalon; their form is not hemispheric but hemi-elliptic. In Stage 7 they progress forward, their rostral pole surpassing the anterior end of the diencephalon, and expand laterally, assuming the shape of real hemispheric vesicles. The telo-encephalic sulcus has become deeper. The anterior end of each vesicle ends in a spur like projection which represents the first rudiment of the rhinencephalon. The unpaired telencephalon is reduced in size. The anterior branches of bifurcation of the telodiencephalic sulcus converge and unite forming the interhemispheric fissure. The paraphysis is thus formed. The author then takes up the much debated question relating to the mechanism of formation of the cerebral hemispheres. The researches of Baer, Reichert, Goette, Mihalcowicz, Remak, Sterzi, Heinrich, Neumayer, Koelliker, His, Minot, Gegenbaur, Hertwig, Kamon are quoted. Luna admits that in *Rhinolophus*, since the epoch of Stage 1, there is a triple subdivision of the telencephalon, without denying that this subdivision may be only apparently due to the presence of the sulci tegmenti. The torus transversus appears first in embryos of 5.5 mm.; it belongs to the subneuroporic plate. The author then discusses the terminology used

by Tandler and Kantor in the constitution of the lamina terminalis. He suggests the name of lamina reuniens be kept to indicate the first, second and third portions of the neural tube which go from the velum transversum to the optic chiasm, and to name the first portion supraneuroporic plate, the second subneuroporic plate and the third preoptic plate, this last portion belonging to the ophthalmencephalon. The rudiment of the striate bodies is formed by a very abundant germinal layer and by a thick mantle layer. The choroid plexuses of the lateral ventricles begin to appear in Stage 7. The appearance of the paraphysis previous to the epiphysis and to the choroid plexuses of the lateral ventricles is a fact worthy of note. The author is not in a position to establish a morphologic correspondence between that bud of the telencephalic roof found in Stage 3 and analogous structures described by D'Erchia, and Chiarugi in *Torpedo ocellata* and by Livini in birds. This formation, he says has a transient existence, because it is not recognizable in the successive stages. The paraphysis therefore, which in *Rhinolophus* appears in Stage 6, is not developed from such a bud.

NACCARATI, New York.

ASTHMA AND VAGO-SYMPATHETIC IMBALANCE. J. GALUP, Presse méd. **31**:555 (June 20) 1923.

The writer treats his subject at length, discussing, together with his own, the clinical and laboratory data and theories of two score other investigators. His ultimate conclusion is that asthmatics, both in and between attacks, must be sufferers from a disturbed equilibrium between the activities of the vagus system and those of the sympathetic system, rather than from a disorder of either system alone. He particularly objects to the conception of asthmatic seizures as mainly vagotonic.

The oculocardiac reflex is specially considered. Data were secured in 152 cases of asthma, with the subject recumbent, a measured pressure on the eyeball of 500 gm., and the pulse counted for one minute. A slowing of more than ten beats per minute was deemed an exaggerated reflex. Thirty per cent. of the cases yielded a normal response; 35 per cent., exaggerated; 35 per cent., inverted or abolished. That is, 70 per cent. of these asthmatics showed an abnormal oculocardiac reflex, but as frequently so in the direction of sympathicotonia as in that of vagotonia. Another author had found the reflex inverted or abolished in over 60 per cent. of his cases.

The relative frequency of this inverted or abolished oculocardiac reflex is noted in quotations from other works, and the conclusion is reached that this phenomenon does have a sympathicotonic significance. The writer finds in comparative researches further evidence of the simultaneous appearance of an inverted oculocardiac reflex with pharmacodynamic reactions of sympathicotonia. He disagrees with Barré, who believes that the oculocardiac reflex is sympathetic-vagal rather than trigeminal-sympathetic-vagal, and that cardiac acceleration will not be the effect of ocular compression but of superadded phenomena, chiefly pain, and will not express a sympathicotonia.

Galup claims that the finding by some authors of a greater incidence of *increased* (i. e., vagotonic) oculocardiac reflexes among their asthmatics, can be explained by making the tests only when the patients were having attacks, and by using unstandardized technic. He distinguishes the equilibrium of an asthmatic's vegetative nervous system in periods when attacks prevail, from that in periods quite free from attacks.

During free periods, the sympathicotonic signs oftenest observed were dermographia, dryness of the skin, impulsiveness, habitual constipation, and increased tendon reflexes. A number of cases were found to show vagotonic oculocardiac reflexes with other signs mainly sympathicotonic, or vice versa. "Vagotonia" and "sympathicotonia" as entities are set aside in favor of the conception of "vegetative dystonia."

The writer further contends against the attacks themselves being explained as an expression of vagotonia exclusively. An attack may follow a paroxysm of hypervagotonia or it may follow a modification of the sympathetic tone, either the latter's inhibition or the succession within its influence of inhibition and excitation. Regarding interrelations between anaphylaxis and vagotonia, while it is true that the serologic phenomena of anaphylactic shock have been exaggerated by pilocarpin and diminished by atropin, this does not disprove that effects could be produced in anaphylactic susceptibility by modifications of sympathetic tone. In the great majority of asthmatics, a condition of permanent imbalance between the vagus and the sympathetic systems seems to prevail.

The probable causes of this imbalance, and of the paroxysms, are discussed separately. Local causes of permanent imbalance are said to be irritations at a point in the organism innervated by the vegetative system (as a whole), irritations which provoke a hypertonic condition, either of the whole system or of one of its antagonistic components. In most instances this irritation appears to be in the respiratory apparatus, often as a pulmonary sclerosis. Statistics are given to show the frequency of an abnormal oculocardiac reflex in various nonspasmodic respiratory disorders.

General causes of the permanent imbalance are found in the physiologic and pathologic variations of the internal secretions, also in infections and intoxications. The state of vagosympathetic imbalance is claimed to favor serologic shock (the hemoclastic shock of Widal and others), and repeated serologic shocks may also enhance the intensity of an existing imbalance, so establishing a vicious circle. Colloidoclastic shock is considered again as the chief cause of the actual attacks of asthma.

HUDDLESON, New York.

THE TREATMENT OF BRAIN ABSCESS BY THE INDUCTION OF PROTECTIVE ADHESIONS BETWEEN THE BRAIN CORTEX AND THE DURA BEFORE THE ESTABLISHMENT OF DRAINAGE.

CHARLES E. DOWMAN, Arch. Surg. **6**:747 (May) 1923.

Dowman calls attention to the necessity for creating adhesions in the subdural and subarachnoid spaces, before free drainage of a brain abscess is attempted. To cause the formation of these protective adhesions, only a small trephine opening is made over the supposed site of the abscess. The dura is opened for no more than a half centimeter and the abscess is probed for with a ventricular needle. As soon as the abscess is reached and a few drops of pus have escaped, the needle is withdrawn slightly until the pus ceases to appear. The exact depth and direction of the needle are then calculated.

The prevention of the free escape of pus at the time of operation cannot be too strongly emphasized, for contamination of the wound may result, and the sudden release of the intracranial tension may prevent the formation of adhesions in the subdural and the subarachnoid spaces.

After the depth and direction of the abscess have been calculated, the needle is entirely withdrawn, and a small filiform drain of folded rubber tissue is inserted down the track of the needle in the direction of, but not into, the

abscess. The wound is then covered with a properly fitted dressing, which is not changed for two or three days. During this time, the rubber tissue wick, acting as a foreign body, stimulates the formation of adhesions between the brain cortex and the dura, forming a well-defined ring around the drain. At the first dressing, the wick may be inserted into the abscess. This usually causes little drainage unless the pus is under great tension. Two days later, the second dressing may be attempted, and a second small rubber tissue wick inserted down the track beside the first. The dressings may then be changed daily, for by this time the protective adhesions are wide-spread and firm enough to permit the gradual enlargement of the drainage track by means of additional small wicks of rubber tissue. Within a week or ten days, it is safe to withdraw these wicks and to substitute a drainage tube (preferably the eye portion of a No. 10 English rubber catheter) down the well formed drainage track into the abscess. Usually, free drainage does not take place until tube drainage is established. The dressings from this time may be performed as frequently as desired, the tube being removed and cleansed if suspected of being clogged with exudates. No undue haste in permanently withdrawing the drain should be practiced. As long as drainage occurs at all, the tube should be kept in situ and only removed permanently when all evidences of the infection have subsided. It is safe to leave the tube inserted for a week or ten days after active drainage has ceased, and gradually withdraw it by daily shortening. It may be two or three months before the drainage tube is dispensed with, although the average case does not require so long.

The author's conclusions are that in this way the danger of meningitis following drainage is minimized, and the small trephine and dural opening prevent the formation of a cerebral fungus.

GRANT, Philadelphia.

TAY-SACHS DISEASE: A STUDY OF THE SYMPTOMATOLOGY AND PATHOLOGY OF THREE ATYPICAL CASES. H. W. TORRANCE, Glasgow M. J. **97**:193 (April), 263 (May), and 341 (June) 1922.

In the first paper this author presents a brief summary of the literature bearing on the nomenclature of this disease. He discusses the symptomatology of the juvenile and infantile types with reference to sixty-seven (personally verified) reported juvenile cases. Torrance calls special attention to the fact that, contrary to the usually understood rule, infantile cases do occur in Gentile families, and juvenile cases among Hebrews. He tries, without reaching a definite conclusion, to find connecting links between the infantile and juvenile types.

In his second paper the author gives the detailed history of three children (one male and two female) in one family, suffering with Tay-Sachs disease, which may be summarized as follows:—the family history was negative; the parents were both Gentiles. Birth in each case was normal; one child was bottle-fed, the other two were partly breast-fed. Growth and development were normal to the end of the first year. The initial symptoms began just after the first year with nystagmus, dental caries, and cessation of attempts at walking and talking, followed by apathy, listlessness, "screaming fits" and progressive mental enfeeblement. On admission to the hospital they showed varying degrees of mental impairment, were irritable and apathetic. Physically they were feeble, unable to stand or sit (except Case 3), the arms were spastic and the legs occasionally so, all tendon reflexes were greatly increased; Babinski's sign was present; sphincter control was lost; nystagmus was present;

the pupils reacted normally to light and in accommodation; and double optic nerve atrophy was present. No macular changes were demonstrable. In Case 1 a patch of pigment was present on the temporal side of the disks; in Case 2 no pigmentary changes were present; in Case 3 pigment was present at the periphery of the fundus at the last examination. Sight and hearing were present in a limited degree. Examination of the chest and abdomen gave negative results in each case. Urinary, cerebrospinal fluid and Wassermann tests were negative. Each child became progressively worse and death was due to bronchopneumonia at 4 1/12, 3 2/12, and 2 5/12 years respectively.

In the third paper Torrance presents details of the gross and microscopic findings in the tissues removed at necropsy. The thoracic and abdominal organs were negative except for the bronchopneumonia. Grossly, the brain gyri in Cases 2 and 3 were wasted and shrunken; the cortex was indurated in the rolandic areas and over the occipital poles. The basal nuclei in Case 2 were small. There was definite atrophy of the cerebellum with narrow laminae and wide sulci. The pons, medulla, spinal cord and optic nerves were diminished in size. The material from the nervous system was fixed in Müller's fluid. Sections from the ascending frontal, the ascending parietal, and the mid frontal convolutions, the angular gyrus, the cuneate lobule, occipital pole, superior temporal convolutions, the basal ganglions, the midbrain, the cerebellum, the pons, the medulla, the spinal cord (three levels), the third to the eighth and the twelfth cranial nerves, the optic nerves and tracts, and the retinae (Cases 2 and 3) were studied. This material showed varying degrees of degeneration of the pyramidal cells of the cerebral cortex, Purkinje cells in the cerebellum, and the large angular cells in the spinal cord; degeneration of the motor tracts in the brain and spinal cord; degeneration of the optic nerves and tracts. Although the maculopapillary bundle was somewhat degenerated it did not seem to be especially involved. The retinal material unfortunately had been fixed in formaldehyd and was not satisfactory for examination, although there was an apparent diminution of the ganglion cells, and in places the inner molecular layer was thinner than the outer layer.

In the bibliography this author gives a list of all published reports on Tay-Sachs Disease.

This splendid and detailed paper should be read in the original by those interested in Tay-Sachs Disease.

POTTER, Akron, O.

CLINICAL STUDY OF A CASE OF PARAPLEGIA DUE TO A BULLET AND COMING ON FOUR YEARS AFTER THE WOUND. J. A. BARRÉ, Encéphale **18**:441, 1923.

In November, 1915, the patient was hit by a bullet which was found point upward in the vertebral canal opposite the eighth and ninth thoracic vertebrae. At this time there were no neurologic symptoms. In September, 1919, the patient experienced a sense of weight in his legs, formication, and a sense of constriction and coldness in the toes of both feet. Constipation was present. Signs of medullary compression appeared and by the end of May, 1920, he was almost completely paralyzed. The reflexes of defense were interesting. In seeking the upper limit of the cutaneous zone from which these reflexes could be elicited it was found that at the inguinal fold there was a latent period of five or six seconds, and that at the level of the umbilicus it was fifteen seconds on the right and twenty-eight seconds on the left. Frequently the movement followed only when the pinching of the skin was interrupted. The author

suggests the possibility of other causes for these movements, but is inclined to believe that cutaneous stimulation was really responsible, since spontaneous movements were lacking.

Objective sensibility was reduced or abolished in varying degrees to the level of the seventh thoracic segment, although there was very little diminution in the sacral distribution.

At operation the bullet was found embedded in a mass of tough fibrous tissue, 6 x 2 cm., arising from the dura and compressing the cord. It was removed without opening the dura. Following operation catheterization was necessary. Five days afterward there were spontaneous contractions of the lower extremities, but these disappeared in a short time and convalescence was rapidly established. A month after operation the patient was able to walk a little without support. Sensation was normal when tested objectively, but the patient complained of paresthesia in his toes. The gait was distinctly spastic.

The patient was reexamined nearly three years after operation. He was able to walk 15 to 20 kilometers without distress and could run, but he noticed that his left foot hit the ground harder than the right and accommodated itself less well to the inequalities of the ground, and also that in hopping on one foot there was some irregularity in alighting in the same spot. He stubbed his left toes more than the right. Visceral function was completely restored. There was some subjective change in sensation at the level of the seventh thoracic segment. The left lower extremity was hypothermic. The tendon reflexes on the left were still more prompt than normal; an abortive ankle clonus and Babinski sign were present. The area for excitation of the reflexes of defense had extended to the sole of the foot.

The points of interest are the long silent period of the bullet, the question of severe constipation as an early sign of medullary compression, of paresthesia far below the level of the lesion and its interpretation, the latent period of reflexes of defense, the residua of the paraplegia, and the prolonged tolerance of the spinal cord in the presence of an "intraspinal" projectile.

FREEMAN, Philadelphia.

ARTIFICIAL NERVE BRANCHES FOR INNERVATION OF PARALYZED MUSCLES. BYRON STOOKEY, Arch. Surg. **6**:731 (May) 1923.

Stookey finds that, if an autogenous graft be anastomosed to a slip from a motor nerve and the free end is inserted in a muscle, regeneration of motor fibers occurs along the graft, these fibers taking up the motor function to the muscle into which they have been implanted. In this way, a muscle in which the motor nerve supply has been cut may be neurotized. He finds further that the same results may be obtained without the use of an autogenous graft, if a slip be dissected away from the side of a motor nerve and inserted into the paralyzed muscle. He believes that the best results are obtained if the slip, or graft, is sutured into the muscle at the point where the motor nerve normally enters it. This, however, is not essential, for neurotization apparently occurs, wherever the nerve graft reaches the muscle. The importance of these experiments cannot be overestimated, for by this method nerve control over a paralyzed muscle may be regained in cases in which, for some physical or technical reason, it has been found impossible to perform effective nerve suture on the nerve trunk.

The technic is simple. Apparently the mere insertion into the muscle of an autogenous graft, or of a slip dissected from the trunk of a neighboring motor nerve, is sufficient to cause neurotization of the paralyzed muscle. Care must be taken to inform the patient that the dissection of the slip from the nerve trunk may cause him motor weakness in the muscles controlled by that nerve.

The author believes, however, that owing to the shifting pattern of the nerve fibers in any given nerve trunk and the possible downgrowth of the new neuraxes to replace those cut in the dissection of the slip, the chance of serious motor involvement is reduced to a minimum. He says nothing, however, of the possibilities of placing under control of a single motor nerve an antagonistic group of muscles. Reeducation is, of course, imperative in these cases.

Protocols of five animal experiments are given. The conclusions are that: (1) When muscular branches are destroyed and nerve suture is impossible paralyzed muscle may be innervated by the formation of an artificial nerve branch. (2) When a free nerve transplant is sutured to the nerve trunk and the distal end is implanted directly into the muscle, the free nerve transplant serves as a conduction path from the nerve trunk to the muscle. (3) An artificial nerve branch may be made for a muscle from a nerve trunk which normally supplies the muscle; or, if this nerve trunk is totally destroyed, a branch may be made from a adjacent nerve. (4) Evidence that paralyzed muscles may be neurotized by an artificial nerve branch was shown by electric stimulation of the artificial nerve branch, resulting in a rapid and quick contraction of the muscle; by the normal size and color of the muscle; and by the histologic findings which revealed normal striations in the muscle fibers. The presence of nerve branches and nerve fibers in the muscle thus innervated is conclusive evidence that neurotization has taken place.

GRANT, Philadelphia.

THE REMOTE EFFECTS OF GUNSHOT WOUNDS OF THE HEAD. L. B. RAWLINGS, Brit. J. Surg. **10**:93 (July) 1922.

This author summarizes the results of four-hundred and fifty-two cases, divided as follows: scalp wounds forty-seven, non-penetrating wounds one-hundred and nineteen, penetrating wounds two-hundred and six, perforating wounds nineteen, fractured base sixteen, decompression forty. Of the forty-seven cases with scalp wounds ten per cent. were incapacitated for work, fifteen per cent. suffered with paralysis and ten per cent. with seizures. Rawlings states that scalp wounds must not be looked on as of no consequence as they are frequently accompanied by contusions of the brain or even hemorrhages. Of the two-hundred and six cases with penetrating wounds of the head thirty-eight per cent. were unable to work, thirty-five per cent. had paralysis, and thirty-five per cent. suffered with seizures. Of the nineteen cases having perforating wounds twenty-one per cent. were incapicated, thirty-two per cent. had paralysis, and sixteen per cent. suffered with seizures. Of the sixteen cases having fracture at the base fourteen per cent. were incapacitated, twenty-one per cent. had paralysis and fourteen per cent. suffered with seizures. Rawlings describes in detail the headaches which were still present in eighty-nine per cent. of this group of four-hundred and fifty-two cases and recommends, in selected cases, subcortical decompression for their relief. Repeated lumbar puncture has not proved satisfactory in the treatment of these headaches.

Giddiness was present in seventy-seven per cent. and nervousness in sixty-eight per cent. of his cases. He remarks that while retained foreign bodies may remain encysted in the brain substance without producing harmful effects, there is always danger of a "flare" with serious consequences. The author describes in detail his method of decompression and gives a synopsis of forty operated cases. He also describes what he believes to be the best method of closing skull defects by the insertion of a double celluloid plate.

POTTER, Akron, O.

THE THERAPEUTIC USE OF TRYPARSAMID IN NEUROSYPHILIS. W. F. LORENZ, A. S. LOEVENHARDT, W. F. BLECKWENN, and F. J. HODGES, J. A. M. A. **80**:1497 (May 26) 1923.

From the results of experimental work it was evident that the drug possessed an affinity for the central nervous system and it seemed possible that the affinity of the drug for these tissues might be utilized for therapeutic purposes. The spirocheticidal action is comparatively feeble, but in the experimental work with the drug there was noted a promptness of recovery from toxic injury, tolerance to repeated doses, a marked tonic effect, and the ability of the drug to induce resolution and healing of syphilitic lesions, even in the presence of actively motile spirochetes, but without increasing the liability to the occurrence of a generalized disease.

The chief clinical interest in this drug lies in its use in the treatment of general paralysis. In the work reported forty-two advanced general paralytic patients and twelve earlier cases were studied. Of the advanced cases twenty-one have been discharged from the hospitals and are working. The earlier cases were relatively acute and were of the extremely agitated type of general paralysis; in these cases the results have been the most pronounced. Seven have fully recovered their normal mentality, have been discharged and are earning their livelihood; five are mentally in condition to earn their living, but, on account of serologic findings have not been discharged. Other types of neurosyphilis were treated effectively with tryparsamid and mercury salicylate. The authors also recommend its trial in syphilitic patients showing a poor state of nutrition, in those beyond middle age and in cases that are "Wassermann fast." When used in dosage of 3 gm., tryparsamid produces no local or general symptoms, either immediately or late, and can be used to advantage in cases in which the patient cannot tolerate other arsenicals.

The drug possesses the power of injuring the optic tract and should not be used in cases showing degenerative changes in the retina.

NIXON, San Francisco.

THE SKIN SIGNS OF VISCEROSENSORY PHENOMENA IN ACUTE APPENDICITIS. EDWARD M. LIVINGSTON, Arch. Surg. **7**:83 (July) 1923.

Livingston finds a constant increase in the sensitivity of the skin over McBurney's point to pain in cases of acute appendicitis. That this is a reasonable claim is suggested by the association of skin areas with viscera, following the segmental distribution of the "cord zones" of the skin, as described by Head. The area involved in the hyperesthesia is bounded above by a line from the umbilicus to the highest point on the right iliac crest, a line carried from this point to the right pubic spine forms the lower side, and a line from the right pubic spine to the umbilicus closes the triangle. Though this triangle does not correspond to any cord zone or zones and does not include the region

of hyperesthesia which is present dorsally, it is of great clinical value. Skin signs within this triangle and maximal at its center constitute confirmatory evidence of appendicitis. When the signs extend definitely beyond its borders upon the anterior abdominal wall or are maximal elsewhere, they are considered negative for appendicitis. Skin signs above the superior line of this triangle suggest cholecystitis, while those below its inferior line suggest right renal colic, and so forth.

The method of applying the stimulus to this area is to give the skin a vigorous twisting pinch of sufficient intensity to produce discomfort on normal skin. Such an uncomfortable pinch becomes exceedingly painful when applied with the same intensity on an involved zone. The basis of the test is the comparison of the slight discomfort on noninvolved skin with the severe pain on involved skin. The essential factor on applying this test is to use a deep grade of stimulus.

One hundred and thirty-six patients were tested, ten of whom did not have appendicitis, but did not have positive skin signs. Seven were not tested. Of the remaining 119, 101 presented positive signs (84.9 per cent.), and eighteen gave negative results. In all negative cases with appendicitis the organ was either gangrenous or ruptured. Skin signs thus elicited and thus localized constitute evidence of value in the diagnosis of acute appendicitis.

GRANT, Philadelphia.

STUDIES OF EXOPHTHALMIC GOITER AND THE INVOLUNTARY NERVOUS SYSTEM. L. KESSEL, C. C. LIEB and H. T. HYMAN. III. A STUDY OF FIFTY CONSECUTIVE CASES OF EXOPHTHALMIC GOITER. L. KESSEL, H. T. HYMAN and H. LANDE, Arch. Int. Med. **31**: 433 (March) 1923.

The object of this study was to ascertain the natural course of the disease process in exophthalmic goiter. No "specific" therapeutic measures were used. The patients were placed under approximately ideal hygienic and psychic conditions. Phenobarbital or wet packs at 75 F. were given for restlessness and insomnia. With the exception of syrup of ferrous iodid, which was given to reduce the neck circumference, no other drug was generally employed. In 72 per cent. of the cases of this group some psychic insult was noted as a precipitating factor and appropriate measures were taken to alleviate such mental trauma. Whenever focal infection was demonstrable, surgery was resorted to with occasional excellent results. As a rule the patients were kept in bed from six to ten weeks and then sent to the country for a month. With such treatment there was a definite arrest of the symptoms with a return to economic and social capacity within six months. The authors accordingly conclude that the spontaneous course of exophthalmic goiter is toward restitution to normal in the vast majority of cases.

VONDERAHE, Cincinnati.

THE COAGULATORY ACTION OF PITUITARY EXTRACT. ALFRED HANNS, MILAN STÉFANOVITCH and VOISLAV ARNOVLJEVITCH, Presse méd. **31**:302 (March 31) 1923.

Exception is taken to the recent statement of R. Feissly (of Lausanne) denying specificity to the coagulating action of pituitary extract. If, taking his theory that its coagulating action is a phenomenon of shock, we admit accelerated coagulation of the blood could constitute a shock symptom, this

hypercoagulability immediately after the injection of pituitary extract would have to be accompanied by other phenomena characteristic of colloidal shock; e. g., arterial hypotension and leukopenia.

Hypotension frequently, but not always, follows the intravenous injection of extract of the posterior lobe of the pituitary gland; among ten subjects, only two gave a definite reaction, four a slight one, and four were quite negative. Similar results were obtained in a series of nine experiments: three definite, four slight and two negative.

Leukopenia can exist, but it also is inconstant: in nine cases, half an hour after the injection, diminutions of 155, 465, 700, 930, were found, counts that could hardly be considered characteristic of hemoclastic shock; in one case there was no leukopenia but an actual increase in the number of white cells; in four cases only were there notable drops in the leukocyte counts, attributable to colloidal shock (diminutions of 1,395, 2,005, 2,170, and 2,480).

A detailed table is given of nine experiments, showing the number of leukocytes before the injection, and half an hour and one hour afterward; behavior of the arterial tension; modification of the pulse, and acceleration of the coagulation. From these data, no relation is deduced between accelerated coagulation, leukopenia and hypotension. Three cases without leukopenia showed the greatest acceleration of coagulation.

The authors conclude that the property of accelerating blood coagulation is a specific chemical character of pituitary extract, independent of that group of phenomena known as hemoclastic shock (capable of being induced by many varying agents), and different from the properties of all other glandular extracts.

HUDDLESON, New York.

STUDIES IN EXHAUSTION: V. HEMORRHAGE. G. W. CRILE, Arch. Surg. **7**:154 (July) 1923.

In continuing his studies of exhaustion, Crile considers the effects of hemorrhage without accompanying physical trauma. He finds that the immediate effect of an acute hemorrhage on the cardiovascular system is stimulation to compensatory activity. If the hemorrhage is protracted, the power of compensation is gradually lost. The brain shows increased functional activity after acute hemorrhage, manifested by an increase in the percentage of active cells and an increase in its temperature. As the blood loss continues, the temperature of the brain decreases and there is a diminution in the percentage of active cells.

The suprarenals show the effect of hemorrhage by histologic changes characteristic of exhaustion; these are seen in the cortex. There is also an increase in amount of suprarenal secretion. The liver shows similar histologic signs of exhaustion.

There is an increase of the hydrogen ion, and concentration of the blood, following hemorrhage. The effects of hemorrhage on the economy, with the exception of the effects on the constituents of the blood itself, are identical with the effects of physical trauma or of any other exhaustion-producing agent. Apparently, a slight hemorrhage is more dangerous to a debilitated patient than a massive hemorrhage to a vigorous subject. There is, therefore, no practical value in the categorical statement that a certain proportion of blood may be lost with safety.

GRANT, Philadelphia.

ALCOHOLISM AND THE BEHAVIOR OF WHITE RATS. XI. THE MAZE-BEHAVIOR OF TREATED RATS AND THEIR OFFSPRING. E. CARLETON MACDOWELL, J. Exper. Zool. **37**:417 (July) 1923.

An attempt was made to control or modify normal inheritance, with particular attention to mental conditions as revealed in behavior. White rats treated with maximal doses of alcohol fumes daily for twenty-eight days before and during training took more time per trial in running a circular maze than did their untreated brothers and sisters. The criteria involving perfect trials give results that tend in the same direction as those given by the data on time, but they are less conclusive and definite. The treatment of the parents as well as the rats themselves caused no more modification in the maze behavior than was found in the rats originally treated, namely, a tendency to retard learning; but this tendency with the small numbers involved is either on or below the borderline of statistical significance. A small but consistent modification of the maze behavior of the untreated offspring from treated parents was found; this appeared more clearly in the criteria from perfect trials than in the time per trial. Untreated rats from parents treated with mild doses showed no difference in their maze behavior from the controls. The following generalization is reached: Alcoholism in ancestors may modify the behavior of untreated descendants.

WYMAN, Boston.

OBSERVATIONS OF THE CARDIOVASCULAR SYSTEM IN THYROID DISEASE. W. J. KERR and GEORGE HENSEL, Arch. Int. Med. **31**:398 (March) 1923.

The authors studied the cardiovascular condition of 181 patients with thyroid disease; 123 of the cases were classified as adenomas and fifty-eight as hyperplasias; no sharp distinction was drawn between toxic and non-toxic types. In 44 per cent. of the cases cardiovascular symptoms were more or less marked. The symptoms usually complained of were rapid and forceful beating of the heart and "palpitation," while vasomotor disturbances, irregularities of the heart, dyspnea and breathlessness were frequently noted; precordial pain and anginal attacks were rare. The cardiac signs and symptoms in toxic adenomas and hyperplasias were found to differ only in degree; the vascular changes were more marked in the cases of hyperplastic goiter. In about one-third of all toxic cases auricular fibrillation or auricular flutter, usually paroxysmal in type, occurred; the authors believe that such cardiac irregularities are more common than is generally recognized and that such paroxysmal attacks explain the periods of palpitation described by many patients. The authors also believe that the state of the circulatory system is the best index to prognosis and to the time and extent of operative treatment. Treatment toward relieving the myocardium is indicated: rest, sedative drugs when necessary, elimination and the appropriate use of digitalis.

VONDERAHE, Cincinnati.

STUDIES IN EXPERIMENTAL TRAUMATIC SHOCK. THE INFLUENCE OF MORPHIN ON THE BLOOD PRESSURE AND ALKALI RESERVE IN TRAUMATIC SHOCK. MCKEEN CATTELL, Arch. Surg. **7**:96 (July) 1923.

Cattell studied the influence of morphin on the blood pressure and alkali reserve in traumatic shock. He found that, in the cat, morphin does not

accelerate the production of shock as measured by blood pressure, and in the normal animal, even in large doses, it caused only a temporary depression of the blood pressure. Morphin, moreover, apparently prevents the depletion of the alkali reserve of the body, which occurs when the blood pressure has been reduced below 60 mm. of mercury. If morphin was injected in large doses, the alkali depletion was not only prevented, but there resulted a return almost to normal in the alkali content of the blood. Injection of morphin in large doses resulted in only a slight and temporary fall in gaseous metabolism. The changes in the gaseous exchange are probably not sufficient to explain the increase in the alkali reserve through a decrease in the oxygen requirements of the tissues.

GRANT, Philadelphia.

HIGH GRADE CHOKED DISKS IN EPIDEMIC ENCEPHALITIS. WILLIAM G. SPILLER, J. A. M. A. **80**:1843 (June 23) 1923.

The diagnosis of epidemic encephalitis may in some instances be difficult; the symptoms may resemble those of brain tumor, and this difficulty may be greatly increased when choked disks are present. The author states that he has seen at least three cases in which the diagnosis of encephalitis seemed definite and yet choked disks were present. Two cases are reported in this paper. Both cases were in boys, one aged 16 years, the other 17.

NIXON, San Francisco.

A CASE OF RHEUMATIC FEVER WITH CEREBRAL SYMPTOMS SIMULATING ENCEPHALITIS LETHARGICA. LEWIS FOX FRISSEL, Arch. Int. Med. **31**:411 (March) 1923.

The onset in this case was characterized by an acute tonsillitis followed two days later by joint pain and erythema nodosum. Seven days later meningeal symptoms developed. At this time there was slight diplopia, doubtful Babinski sign and slight stiffness of the neck. Lumbar puncture revealed two cells and a faintly positive butyric acid test. The central nervous system signs increased in intensity; the patient became semistuporous and the face assumed a parkinsonian mask-like expression. About the second week there were slight choreiform movements and slight facial weakness with marked somnolence. A blood culture taken during the height of the illness was negative. About the third week the patient began to show signs of improvement and subsequently a complete recovery was made.

VONDERAHE, Cincinnati.

SYPHILIS AND MENTAL DEFICIENCY. G. J. KEY and A. PIJPER, So. African Med. Rec. **20**:8, 142 (April 22) 1922.

These authors present the report of an investigation of the Wassermann serum reaction in 217 cases of mental deficiency. The estimation of the degree of the mental defect was based on the result of intelligence tests (Stanford revision of the Binet-Simon scale). They report thirty-nine definitely positive and twenty weakly positive reactions among ninety-two idiots; thirty-two definitely positive and eleven weakly positive among ninety imbeciles; eleven definitely positive and five weakly positive among thirty-four feebleminded children. One case of juvenile general paralysis with a positive reaction is

included in this series. In thirty-five of the cases showing some form of paresis (diplegia, hemiplegia or paraplegia), nineteen gave a definitely positive and nine a weakly positive reaction. In studying the results of this series in relation to age incidence the authors note that, after the age of 25 years, the relative percentage of definitely positive results decreases while the percentage of weakly positive results increases.

POTTER, Akron, O.

THE ARSENIC CONTENT OF THE SPINAL FLUID AFTER THE INTRAVENOUS ADMINISTRATION OF SILVER ARSPHENAMINE. (A CLINICAL AND BIOCHEMICAL STUDY OF NEUROSYPHILIS. PART I.) LEON H. CORNWALL and C. N. MEYERS, Am. J. Syphilis **7**:287 (April) 1923.

Cornwall and Meyers give the results of 239 spinal fluid examinations of 151 patients receiving silver arsphenamin. In 78 per cent. of the cases arsenic was present in sufficient amount for quantitative determination. The arsenic content in general tended to fall after the first two hours. It rose slightly between twenty-four and forty-eight hours and at the end of seventy-two hours was present in still greater quantity. This latter characteristic, however, the authors point out, is not equally true for all types of neurosyphilis.

VONDERAHE, Cincinnati.

CALCIFICATION AND OSSIFICATION OF THE MENINGES. ALBERT E. HALSTEAD and FREDERICK CHRISTOPHER, Arch. Surg. **6**:847 (May) 1923.

Halstead and Christopher report a case of calcification and ossification of meninges diagnosed by roentgenogram. From a review of the literature and their experience in the case reported, they conclude that: (1) Ossification and calcification of the meninges are not rare. (2) Areas of ossification may arise from (a) the retention of the osteogenetic function of islands of dura; (b) an inflammatory process. (3) Areas of calcification may arise as: (a) the result of a not far distant absorption of calcium of the skull and its subsequent deposition in the meninges; (b) the result of an inflammatory process. (4) In cases of suspected brain tumor, the demonstration by means of the roentgenogram of calcification of the meninges is additional evidence of the existence of a brain tumor exerting a circumscribed pressure on the skull. (5) The chance finding of ossification or calcification in roentgenograms of the head affords occasion for an intensive neurologic study of the case in question.

GRANT, Philadelphia.

TREATMENT OF MIGRAINE WITH PEPTONE. EVIDENCE OF THE ANAPHYLACTIC CHARACTER OF THE SEIZURE. J L. MILLER and B. O. RAULSTON, J. A. M. A. **80**:1894 (June 30) 1923.

Twenty-five patients were treated for migraine by the intravenous injection of peptone; as the result of this treatment, 36 per cent. were much improved, 48 per cent. were moderately improved, and 16 per cent. were not benefited. Whether these results were due to desensitization or to some other action of the peptone is not known. Clinically, migraine has many of the characteristics of a sensitization disease.

NIXON, San Francisco.

MONGOLISM IN ONE OF TWINS AND THE ETIOLOGY OF MONGOLISM. T. HALBERTSMA, Am. J. Dis. Child. **25**:350 (May) 1923.

The author reports five cases of twins in which one of the infants showed evidence of mongolism. In three of the cases both sexes were represented; in the other two cases, in which the twins were of the same sex, each child had its own placenta and membranes, so that in all instances a two egg pregnancy was demonstrated. The occurrence in one twin is pointed out as indicating a germinal rather than an acquired origin of the condition; accordingly, the author believes that the basis of mongolism is a defective germ plasm.

VONDERAHE, Cincinnati.

PRESERVATION OF THE FACIAL NERVE IN THE RADICAL TREATMENT OF PAROTID TUMORS. ALFRED W. ADSON and WILLIAM O. OTT, Arch. Surg. **6**:739 (May) 1923.

Adson and Ott describe a technic for the location of the facial nerve and its preservation in dissections about the parotid gland. They find that with their technic, they have been able to preserve the facial nerve in cases in which it was necessary to remove tumors from the parotid, or even to extirpate the entire gland.

GRANT, Philadelphia.

Society Transactions

NEW YORK NEUROLOGICAL SOCIETY

Regular Meeting, October 2, 1923

E. G. Zabriskie, M.D., *President, in the Chair*

A CASE OF DYSTONIA WITH ATHETOID FEATURES. Dr. I. Margaretten.

This patient has a sequential history since birth which has a definite bearing on the etiology of her disease. Birth was normal from a tuberculous mother. The family history was otherwise negative. Two weeks after birth she had convulsions and developed a right hemiplegia. She cried almost incessantly for two years. She received a trauma to the head at two years of age and shortly after this her aunt, with whom she lived, noted peculiar movements in the right upper and lower extremities. A year ago the same type of movements developed in the left upper and lower extremities. Today she cannot walk or sit up without assistance; nor can she talk beyond a few monosyllabic words.

Physical Examination.—This reveals a poorly nourished child, with apparent dystonic and athetoid movements, most intense on voluntary effort, when ataxia also becomes evident. The right upper and lower extremities still show definite weakness although there is no sign of atrophy or fibrillation anywhere. The abnormal movements are less evident in the muscles of the trunk and those supplied by the cranial nerves. They are most marked in the face and tongue and least in the eyes. Voluntary movement improves with continued effort and there is a tendency even to associated automatic movements in walking. There is alternating hypotonia and hypertonia. There is no deformity or contracture. The emotion exhibited is pleasant even with painful stimuli. There are no disturbances of reflexes, sensation or cranial nerves beyond these adventitious movements. The mental state seems to be fair, if allowance is made for her physical handicap.

TWO JUVENILE CASES OF MYASTHENIA. Dr. M. Neustaedter.

Case 1.—R. S., a girl, aged 8½ years, was born in the United States of Jewish parents who were second cousins. There is a history of tuberculosis in the family of her father; otherwise the family history is negative so far as could be elicited. The child was born at full term after normal labor, and there was no oligopnea. She was breast and bottle fed; teething began at 7 or 8 months and was accompanied by slight fever; she sat up at 6 months, walked at 16 months and talked at 1 year. She had pertussis and measles without any sequelae. Tonsils and adenoids were removed three and one half years ago. She coughed constantly from birth up to the age of 3 years and was habitually constipated up to the age of 2.

At the age of 6 months the mother noticed that she could not open her eyes as completely as her normal brother. Only on rising after sleep would she open them completely and keep them open for about an hour. At present

she keeps them open for three hours after a night's rest. During the first year of life she suffered from frequent attacks of dyspnea and at every attempt at vomiting she became cyanotic for a few seconds. She was always weak on her legs and never ran with the ease of her brother. At the age of 4 years her mother noticed that, after walking a few minutes, she became tired, was unable to proceed and had to be carried. At present after walking two blocks she complains of pain in the legs and is forced to rest. She cannot walk up stairs without holding onto something. Her arms are fairly strong, but, after writing for about an hour, she is forced to stop on account of weakness. If pushed slightly she falls.

Her school teacher reports as follows: "Ruth falls when she plays running or skipping games; she has difficulty going up and down stairs; every exercise seems to tax her strength so that she appears used up after any exercise; even in marching she lags and tires. In school work she does well. In reading and writing she shows some nervousness and her voice is nasal in reading."

Physical Condition: Blood pressure is 70 systolic, 50 diastolic; pulse 108 and small; thoracic and abdominal viscera are normal; pupils are sluggish to light; accommodation cannot be tested for she has a complete ophthalmoplegia. There seems a slight mobility of the eyeballs downward. The ocular fundi show contracted arteries and injected veins, the disks are normal. There is myopia. Superficial and deep reflexes are present. There are no sensory disturbances, the motor power is fair and there is no adiadokokinesis, but there is hypotonia in all muscles and they tire easily on passive movement. There is considerable weakness in all facial muscles, especially in the orbiculares palpebrarum and frontales. The response to the faradic and galvanic currents is normal, but there is a distinct myasthenic reaction in all muscles of the extremities, which is absent at times after the administration of nux vomica for a few weeks.

Case 2.—L. G., a girl, aged 10 years, was born of American parents. There is a history of tuberculosis on both sides of her family and diabetes and neuropathy on her mother's side. "Lots of rheumatism" on both sides. The child was born after a very difficult labor, rather late in the life of her mother. She was breast fed for two months and then bottle fed; teething began at 10 months, walking at 16 months and talking at one year. Between the second and sixth week of her life she had three convulsions that lasted several minutes. She had measles and influenza without sequelae. Tonsillectomy was performed two years ago. She suffers from chronic constipation. In school until two years ago she did good work. She is left handed.

Two years ago she awoke with a peculiar cry and had an epileptic seizure of the grand mal type lasting several minutes. When out of the attack she vomited. These seizures came on mostly during sleep at intervals of from five to ten days until a year ago when she became subject to daily petit mal attacks; only one grand mal seizure has occurred since February of this year. She bit her lip several times during the attacks and urinated involuntarily. There is history of trauma to the head four weeks before the onset of the first attack.

Early this year she began to experience difficulty in chewing and swallowing so that only semisolid food could be given and at times only in the recumbent position. At the same time she had difficulty with speech, being unable to finish words, dropping the last syllable, until at the end of February this year she

became completely aphonic. At this writing the mother informs me that one morning she said a few words with difficulty; she is beginning to chew solid food and swallows better.

Physical Condition: Blood pressure 80 systolic, 50 diastolic; pulse 96 of good volume; thoracic and abdominal viscera normal; pupils respond promptly to light and accommodation; the ocular fundi show the veins injected and pallor of the temporal side of the disk in the left eye; the pharyngeal reflex is absent; all other superficial and deep reflexes are present; there are no sensory disturbances and no vocal cord paralyses; she protrudes the tongue with some difficulty and is unable to raise it to the roof of the mouth; on attempting to move the tongue, a considerable tremor sets in; there is flattening of the right facial muscles and she is unable to coapt the lips tightly; saliva is constantly drooling; there is complete aphonia. There is normal response to both electrical currents in all facial muscles. The myasthenic reactions could not be tested for. There is no disturbance in any of the other muscles.

Comment.—In both of these patients the blood Wassermann reaction is negative, and there is no deviation from the normal in blood picture or chemistry; urines also are negative, and roentgen rays show no thymus shadow or other abnormality.

The interesting feature in Case 1 is that the affection seems to have appeared before the first year of age. The earliest case on record began at the age of 2½ years, and was reported by Mailhouse. The second case came on quite early in life and was accompanied by so-called essential epilepsy. So far as I could discover in literature there is no case recorded associated with epilepsy.

DISCUSSION

Dr. L. Pierce Clark: Have you studied the recent literature with the idea of finding out the frequency of convulsions in these cases?

Dr. Neustaedter: I believe these are two separate and distinct phases. I do not think the myasthenia has anything to do with the epilepsy. I think it precedes the condition.

Dr. L. Pierce Clark: I have never seen a case as young as this and have never seen convulsions mentioned. I have been over the literature completely. You say the roentgen-ray examination in this case showed nothing; has any chemical examination been made?

Dr. Neustaedter: In a few cases acidosis is found, but nothing important as regards creatinin. The question in these cases is what is to be done. Dr. Dana suggests massive doses of strychnin, but I am afraid of large doses. I have used nux vomica, and this does some good; it suspends the myasthenic reaction.

Dr. I. Abrahamson: Myasthenia gravis at 6 months of age is very rare; the persistence of the signs and symptoms without remissions is rare; other features are rare; too many exceptions to the rule are not apt to be present in one case. The question of infantile nuclear aplasia or progressive nuclear disease must be borne in mind. On the facts presented, further observation is necessary before a definite diagnosis is made. The other child is a very peculiar case especially in regard to the sudden and complete aphonia.

Dr. M. Neustaedter: I said that the aphonia was gradual, preceded by partial aphonia, or clipping of words. I have had her in the hospital for eight days, on a mixed diet. She has had one petit mal attack without loss of consciousness. During that time I tried to persuade her to talk but without

result. In regard to the girl with ptosis, I thought first of hypoplasia of the third, fourth and sixth nuclei. I have seen a congenital double facial paralysis which was of nuclear origin. But here you have the muscles of the legs and arms, giving the full myasthenic reaction again and again. I think we must rule out hypoplasia of nuclear origin. This case is practically at a standstill, but there are similar cases in the literature. In the picture taken at 8 months, the ptosis did not show, but the mother explained that the child had just been awakened from sleep and ptosis was not present shortly after sleep.

DR. T. K. DAVIS: I would like to question the diagnosis in Case 2. I think the occurrence of the grand mal and petit mal attacks would at once raise a doubt. Is there any variability in the paresis of the tongue? Does this vary as does the weakness of myasthenia?

DR. M. NEUSTAEDTER: The paralysis of the tongue is progressive; she is unable to lift the tongue. She has a slight facial paralysis. She is unable to close the lips and has difficulty in swallowing which is progressive. The aphonia does not suggest a nuclear origin although some myasthenias begin that way.

DR. T. K. DAVIS: That looks to me like pseudobulbar paralysis, and not myasthenia.

DR. M. NEUSTAEDTER: There is involvement of the seventh and twelfth nerves and of the motor nucleus of the fifth. What etiologic factor would take in three peripheral nerves so far apart? These cases were described by Oppenheim as bulbar paralyses without anatomic findings.

SOME UNUSUAL FORMS OF PSYCHOGENIC EPILEPSY. DR. L. PIERCE CLARK.

There are few nervous diseases so unique in symptoms or pathology as to warrant the statement that they have no anomalous forms. Essential epilepsy is no exception. Some of the names that have been given to the variable and irregular group of essential epilepsies are "emotional epilepsy," "hystero-epilepsy," "affective epilepsy," and "paraepilepsy." The chief significance of these irregular types is that they possess ethical perversions in personality makeup that are so glaring as to overpower the picture of infrequent seizures. Secondly, they generally have a favorable prognosis so far as riddance of the convulsive phenomena is concerned; but there is some doubt as to whether the ethical perversions do not continue and gradually pass into specific and enduring antisocial deterioration.

The outstanding syndrome as the basis of the unclassified type is undue and labile affectivity. In most instances the seizure phenomena are not essentially dissimilar to those seen in essential epilepsy, from which they are differentiated only with the greatest care, not on the basis of the type of the seizure but of the antecedent or subsequent mental state. Unfortunately, both for physician and patient, there are fewer of the so-called affect epilepsies than one would wish to suppose. They are essentially to be characterized by (1) psychogenetic factors; (2) absence of the makeup of the essential epileptic as well as his enduring mental stigmata (deterioration); (3) intractability to sedative therapy; and (4) response to psychotherapeutics, analysis, and training.

In consideration of the affective epilepsies in childhood, obviously the personality makeup of the essential epileptic is largely wanting. Symptoms of many of these young patients disappear spontaneously or are early outgrown

under the physician's or pediatrist's care, so neuropsychiatric studies are not frequent. Perhaps the observer who has most thoroughly studied these peculiar types in children is Stier. The condition is not so infrequent, however, as his observations would seem to imply, but it may be diagnosticated incorrectly either as hysteria or as a condition of spasmophilia of childhood. The state does not constitute a distinct clinical entity. Some of the children are obviously suffering from the beginnings of essential epilepsy and after a more or less brief pause continue an epileptic career; others have a mixture of symptoms not so very different in general outline from anomalous spasmophilics; while still others are not essentially dissimilar to attacks of anger, or to "opportunity convulsions," which closely ally them to some of the protean forms of hysteria of childhood and early adolescence. A few would seem to be but a very early form of the affect group and not unlike the Westphal type of affect epilepsy. Many of the latter also in turn shade into essential epileptics on the one hand and pure psychopaths on the other. Finally, the material in the affect group will also prove not very dissimilar in many instances to the anomalous material of dementia praecox and epilepsy in which it is a nice question whether the latter exists as an association disease or as an atypical form of dementia praecox on the one hand and an anomalous essential epilepsy on the other.

In the entire service at Manhattan State Hospital, under institutional care at the present time, I found but a dozen cases of dementia praecox who had had one or more convulsions. Without exception there was an absence of the epileptic makeup in these cases and none came within the range of the epileptic type. The ages of the patients ranged from 22 to 50 years. The makeup was typically that seen in the dementia praecox of paranoid and hebephrenic varieties. In the cases in which but one attack occurred, in one patient the seizure was described as a dizzy spell or faint as the result of being overcome with the heat; in another there was a convulsion in which the patient became purple and foamed at the mouth. One male patient fell from a tree at 11 years of age when he became unconscious for a few minutes, but he made good recovery from his physical injuries after six months; he had previously had convulsions at the age of 4½ years. Another male patient had nocturnal convulsions at the age of 8 years which continued to the age of 14; he is now 28 years old and there have been no convulsive phenomena for fourteen years. Another male patient, aged 34, had convulsions at 14 averaging one every six months, the last occurring three years ago; his present makeup is dull and apathetic and he has catatonic states. One man, aged 40, had convulsions which were purely specific; he contracted syphilis at 19. In the remaining cases studied, the convulsions came within the range of spasmophilia of childhood.

In conclusion we may say that the so-called affect epilepsies in the adult prove most frequently to be really psychoneuroses, or protean forms of hysteria. The respiratory affect convulsions of childhood need our continued study to eliminate them from the beginnings of essential epilepsy. This task is both a pediatric and a neurologic one. Finally, the supposed occurrence of discharge convulsions in dementia praecox is much rarer the more definite the syndrome of dementia praecox is made. It seems unlikely that there is an association disease of dementia praecox and epilepsy. The disease entity is either one or the other. True epileptic attacks are rare outside of the essential type except in organic brain disease or as an occasional accompaniment of internal disorders such as diabetes, nephritis, and the like.

DISCUSSION

DR. M. W. RAYNOR: I have been much interested in Dr. Clark's paper and especially in his study of the various types of convulsive phenomena in epilepsy. I feel that this is a real contribution to the subject of epilepsy. I cannot recall a single instance in which a true epileptoid seizure occurred in dementia praecox in my several years experience. There have been syncopal attacks but not true convulsions. I have, however, seen a few patients with manic syndromes who have later developed epileptic convulsions.

DR. L. PIERCE CLARK: I had hoped to get some of the pediatricians here, as I have obtained most of my clinical material from them, especially from Dr. Kerley. I think it is important for the relief of the parents that this group of cases be taken out of the category of true epilepsy. I believe it is important for the pediatrist to cooperate with the neurologist in these cases. In regard to the praecox types, I failed to find an association of the two disorders in this clinical material. Kahlbaum and others have confused hyperkinesis with epileptic phenomena, and this should be corrected. I have seen one patient, a man of 29, a dangerous type, with paranoia. The sensorium was clear but he had two or three periods of typical epileptic convulsions of the grand mal type. These were temporary and have not recurred. It is preposterous to assert, as do some workers, that the association percentage of epilepsy and dementia praecox runs as high as 25 per cent.

IN MEMORIAM

The Secretary, Dr. Atwood, presented the following resolution:

It is my painful duty to announce the death of DR. PAUL WATERMAN, of Hartford, Conn., a member of the New York Neurological Society, who died July 31, 1923, of acute dilatation of the heart, after a strenuous career. He was only 46; born at Westfield, Mass., December 17, 1877. He obtained the degrees of B.A. at Williams, and M.D. at Cornell. After general hospital experience at Bellevue he was assistant resident alienist there for several years and then studied a year in Vienna, becoming, while there, president of the American Medical Association of Vienna. After his return to this country he practiced neurology and psychiatry at Hartford, Conn., and held many positions of honor and activity in hospitals and medical societies, and in the militia of Connecticut. In 1915, he was graduated from the Army Field Service School for medical officers at Fort Leavenworth, and for four months in 1916 was with the federalized Connecticut troops in Arizona on the Mexican border. He joined the regular federal service in 1917 and became assistant division surgeon, 26th division, under Gen. Edwards. In France he was made medical liaison officer of the Division at the headquarters of the Eleventh Corps d'Armée and held that post from February 11 to March 15, 1918. Then, after serving as assistant to the chief surgeon of the Yankee division, he became commanding officer of the One Hundred and First Sanitary Train, which played an important part in much of the heavy work of the division. On October 1, 1918, he was assigned division surgeon, Fourth Division, and on the day of the Armistice commissioned lieutenant-colonel. On March 5, 1919, he was made colonel, and was honorably discharged from the U. S. Army, August 26, 1919, after twenty-one months in France. His localized service in France comprised the Chemin des Dames Sector, La Reine Sector (with three actions), Pas Fini Sector (near Chateau-Thierry), Champagne-Marne Defensive, Aisne-Marne Offensive, Rupt Sector (near St. Mihiel); St. Mihiel Offensive; Troyon Sector; Meuse-

Argonne Offensive; and with the Army of Occupation. After his return to the United States, Col. Waterman continued to live a strenuous life and was loaded with honors, medical, civic, and military, in the State. He was a member of twenty-three societies, clubs and associations, and wrote several papers, chiefly on intracranial injuries and on sanitation. He maintained his membership and interest in our own society for the twenty years he was a member, although unfortunately his distant residence prevented his attendance.

I move that suitable resolutions of sympathy and respect be drafted by a committee appointed by the president to be sent to the widow and a copy kept in the archives of the society. (The motion was duly seconded and carried.)

CHICAGO NEUROLOGICAL SOCIETY

Regular Meeting, October 18, 1923

JULIUS GRINKER, M.D., *President, in the Chair*

PARALYSIS OF LANDRY'S TYPE IN AN ADULT FOLLOWING A BITE BY A WOODTICK. DR. PETER BASSOE. This report will appear in full in a later issue of the ARCHIVES.

EARLY CONVULSIONS IN EPILEPTICS AND IN OTHERS. DRS. HUGH T. PATRICK AND DAVID M. LEVY.

A study of the prognosis of early convulsions was made by comparing those occurring in a group of 500 epileptics with those in a group of 752 "unselected" infants and children. The epileptics were private patients with idiopathic epilepsy and no other clinical findings. The unselected group consisted of infants and children seen at "Better Baby Conferences" in one urban and two rural communities in Illinois. Studies of the occupational classification of the parents of the unselected children showed that this group was representative of "private practice," as contrasted with institutional or dispensary groups. In previous studies the frequency of early convulsions in epileptics was estimated largely from institutional and dispensary records. Such data suffered because the institutional epileptics are largely feebleminded and of low social status, and present a different picture from that of epileptics seen in private practice.

Ninety-eight cases, or about 20 per cent. of early convulsions were found in the epileptic group; thirty-two cases, or about 4 per cent. in the unselected group.

Analysis was made of the validity of the figure "20 per cent." as a percentage frequency for the epileptics. This frequency was, in a general way, inversely proportionate to the age at the time of the first epileptic seizure. The records show that a history of early convulsions was elicited three times as frequently when the epilepsy began in the first decade of life, as when it began in the third decade. A determination of frequency, based on the time elapsing between the first epileptic attack and the time of our first office examination, showed that for the early epileptics there was little difference in the percentage of early convulsions, as shown by the history, whether they came to the office five or fifteen years after the first attack. The difference in the frequency of early convulsions elicited from early and late epileptics is thought to be due largely to the factors of memory, or incompleteness of data due to the mother's

absence at the time of history taking. The difference most likely was not due to something in late epilepsy essentially different from early epilepsy. Based on the instances of convulsions in early epileptics, the frequency of early convulsions in all epileptics is more probably 40 than 20 per cent. The presence of early convulsions *per se* multiplies the individual's ordinary chances of epilepsy by at least five—in itself a scarcely insignificant risk.

There is no definite "interval of safety" beyond which epilepsy will not occur. In our series an appreciable percentage of cases occurred ten, twenty, and even thirty years after the early convulsion. This is quite contrary to the belief expressed by some writers that there is a period of immunity beginning five to ten years after the early convulsions.

Our statistics show a marked contrast between the epileptic and the nonepileptic series of early convulsions in regard to: (1) number; (2) age; (3) type, and (4) assigned cause.

There is a preponderating number of single early convulsions in the nonepileptic group (65 per cent. of the total) and of multiple convulsions in the epileptic group (76 per cent. of the total).

The majority of early convulsions in the nonepileptic group (75 per cent.) occur between the ages of 6 and 17 months, and most of these are between 11 and 13 months. In the epileptic group 63 per cent. of the early convulsions occur under 6 or over 17 months; only 37 per cent. between 6 and 17 months.

As contrasted with the "epileptic" early convulsions, the nonepileptic are typically brief, generalized, and not followed by confusion or prolonged stupor.

The same type of assigned causes occurs in both series with teething highest in frequency in the nonepileptic; trauma, especially birth trauma, and "reflex" causes in the epileptic. In the latter group there is a contrasting high percentage of "unassigned causes."

From these findings it is concluded that the more likely forerunners of epilepsy are convulsions (1) occurring before 6 or after 18 months of age; (2) multiple; (3) severe; (4) localized; (5) assigned to birth trauma, "reflex" causes, or to no cause, with (6) a family history of epilepsy or similar convulsions. The more fully these conditions are fulfilled, the more likely is epilepsy to follow. The most significant are unilateral convulsions occurring after the fourth year and generalized convulsions lasting over half an hour and followed by confusion of torpor for several hours. Especially benign appear to be "teething spasms" occurring between 11 and 13 months.

Our tables indicate a similar distribution of sex (about 60 males to 40 females), in the two groups.

Within the unselected group, the cases with early convulsions contained relatively seven times as many families with instances of early convulsions in relatives as did the remainder, and a much higher frequency of epilepsy.

These studies indicate a similarity in epileptics and in the nonepileptics with convulsions as regards sex distribution and family history of epilepsy. The nonepileptic group without convulsions were relatively free from epilepsy and early convulsions in the family history, as contrasted with those having early convulsions alone. No significant difference was found in the frequency of miscarriages, still births, deaths in infancy, or forceps deliveries between the normal and the epilepsy group. When forceps were commonly used in a community, the number of early convulsions was no greater than in communities in which they were rarely used.

(This article will appear in full in THE JOURNAL OF THE AMERICAN MEDICAL ASSOCIATION.)

DISCUSSION

DR. HUGH T. PATRICK: As this is really Dr. Levy's work, I feel free to state that I believe it to be the best report yet produced on this subject. In this connection two things have interested and surprised me. First, the enormous difference of opinion, as shown in the literature, regarding the significance of infantile convulsions and their relation to epilepsy. Second, I have found that a great many physicians, and even pediatricians, look on infantile convulsions as trifling affairs. For a long time I have thought this to be a mistake, but the investigations of Dr. Levy seem to have converted my impression into a certainty. Furthermore, I believe that many cases considered rather lightly simply as recurrent infantile spasms are really cases of incipient epilepsy and surely should be carefully and intensively treated as such.

DR. PETER BASSOE: I rise only to express my admiration of Dr. Levy's work. My experience with 200 cases (published in *Medical Clinics of North America*, 1923) taught me that this does mean a great deal of work. I would warn against paying much attention to the small number of cases I had. I took epilepsy in a very loose and broad way and included many cases that Dr. Levy quite rightly excluded. There is one problem that I wish Dr. Levy or Dr. Patrick would touch on and that is spasmophilia—just where to draw the line between spasmophilia and epilepsy in infants. It has often been difficult for me to decide whether an attack was really epileptic or whether it was simply spasmophilic. As we know, there is much difference of opinion among pediatrists as to the relationship of spasmophilia to epilepsy. Some say that there is no relation and some say there is. For instance, Morse and Thom, using largely the same material, arrive at different conclusions. Morse found that spasmophilia was apparently unimportant and rarely led to epilepsy while Thom found that it did. It is surprising to me how little real definite information is to be had when you think of the millions and millions of epileptics that have lived and the tens of millions of children who have had convulsions; yet statements are based more on general impressions than on definite data. It is remarkable that more statistics of this kind are not obtainable. All interested in this work should go over their own cases and put them on record. I tried to do that in a small way. If everyone would do this, it would not be long before we would have hundreds of thousands of cases from which to draw conclusions.

DR. DAVID M. LEVY (closing): I am glad that Dr. Bassoe brought up the question of spasmophilia. The prognosis of spasmophilia in regard to epilepsy is still a disputed point as shown in the literature. In our normal series there was no way of ruling out spasmophilia since we saw only one half of the children in our normal series. The number of attacks is usually regarded as a definite point in eclampsia and spasmophilia. If this is a good guide, there were no spasmophilics in our series. The second series of normal cases reported, contained only one case in which there were several hundred convulsions in a day. A number of the so-called teething spasms will, no doubt, be regarded by some pediatricians as evidence of spasmophilia. It must be emphasized that even if this should be true, the prognostic inferences from this study are not affected because we are here dealing with certain facts in sequence, which follow regardless of how they are interpreted. For example, it has been found that those convulsions occurring between the ages of 6 and 17 months, largely attributed to teething, are the least likely to be the forerunners of epilepsy. This

fact follows from the statistical analysis. If it should be found that they are falsely ascribed to teething spasms, but are definitely caused by some other condition, the facts nevertheless remain. We do not hold, of course, that the assigned causes are causes in any sense. They are antecedent or coincident events and the facts warrant the conclusion that they have a definite relation to prognosis.

A MEMORIAL TO DR. JAMES A. KIERNAN. Dr. Harold N. Moyer.

Book Reviews

DIE HIRNKARTE DES PARALYTIKERS. STUDIEN UBER DAS WESEN UND DIE AUSBREITUNG DES PARALYTISCHEN PROZESSES IN DER HIRNRINDE. Von Prof. Dr. Shigeyoshi Saito, (Nagasaki, Japan). Arbeiten aus dem Neurologischen Institute von Obersteiner und Marburg. Vol. 25. Pages 182, with 21 plates. Leipzig und Wien: Franz Deuticke, 1923.

From Lissauer to Kaes there was a tendency to consider general paralysis a system disease involving largely the second and third layer of Meynert. Schaffer accused not so much specific layers but rather the association areas of Flechsig. Nissl and Alzheimer turned their attention more to the histologic process and they and Noguchi brought about the collapse of the notion of toxins and a system disease. Spielmeyer claimed the existence of both factors. Bielschowsky, however, considered the changes of glia and vessels of the degeneration patches as inflammatory and not purely parenchymatous, and Jakob found spirochetes in these foci. All this does not, however, discourage further efforts to correlate the localization and the symptoms, such as were undertaken by Wada for dementia praecox, 1910, (second and third Meynert layer) and since by Josephy (third and fifth layer). Vogt also has furnished a few examples. Saito aims to be more systematic, takes most of the Brodmann areas and studies seven cases, but unfortunately with very meager clinical records (pages 45-48). It is regrettable that the descriptions are cut up according to the localities and that there is no summary according to the individual cases or even according to the regions studied. There is, however, a general discussion of the various tissue elements.

Saito finds the depth of the normal cortex too variable to prove more than a slight reduction in general paralysis. The participation of the pia shows that, throughout, the process appears focal, with very small foci becoming confluent. The relation of the pia to the underlying brain substance shows in only 12 per cent. of the cases the direct transition of an infiltrate into the molecular layer along bloodvessels, with direct penetrations. The pia change is probably not the primary factor, but the cortical process appears to be the decisive factor in the penetration.

The lateral surface shows more involvement of the pia than the mesial surface. But within the lateral regions there is hardly any preponderance of special areas (except perhaps Brodmann's areas 45 and 38). The striking feature is the discontinuity of the process and the impossibility of a temporal correlation of the clinical phenomena with the pial changes.

The next point is the cell alterations: The three types of changes all begin with swelling; one is a coagulative necrosis or sclerosis, the second a "colliquation necrosis" (Nissl's profound alteration), the third similar to the axonal alteration and the acute alteration (two really very different changes!). They occur usually in mixture, the sclerosis preeminently in the second and third layer of Meynert, the swelling more in the deeper layers. The cell deficits tend to occur in groups. All regions may be affected, but in the main the frontal most frequently and the olfactory regions and those nearest the callosum least. The

fibers suffer diffusely (especially the supraradiary and interradiary nets) and in patches, sometimes little in regions with very severe involvement of the pia. The patches are said to produce little tissue alteration and no obvious secondary degenerations; "hence a process similar to multiple sclerosis." The crest of the convolution is apt to be best preserved. The cell and fiber changes do not usually go parallel except perhaps in the diffuse atrophy. A relation to paralytic attacks could not be proved.

The neuroglia shows increase of cells in the acute cases or stages and more increase of fibrils in the chronic cases, and occasionally mixtures. There is little evidence of neuronophagia and a striking tendency to severe damage to the neuroglia cells themselves. The dysplastic glia cells, some of which he calls vesicular cells, are said to occur especially in the second and third layer, and, like the rod-cells, more in the median areas of the hemisphere near the callosum, and least in the sensory and motor cortex and more frequently in the frontal and temporal cortex. Saito considers their appearance as "the mobilization of the last reserves of glia cells for the fulfilment of the functions of the glia."

Acute vascular infiltration cannot be used as a measure of the duration of the process, as it occurs in all cases, even chronic ones. Saito considers it a definitely inflammatory exudation from the vessels into the tissues. The transition between inflammation and degeneration seems gradual but the relations are left uncertain. Marburg suggests that "the individual focus is always acute, but the total disease chronic." Bielschowsky and Jakob have furnished evidence of an inflammatory affection even in the apparently degenerative areas, but according to Saito the proof cannot always be brought. The meningeal infiltrations and the cortical infiltrations are not always parallel. The calcarine cortex is practically free, the sensory and motor cortex relatively so, while the mesial cortex around the corpus callosum is always much involved.

With regard to the correlation of the clinical and anatomical facts, Saito finds in an essentially dementing case more involvement of the projection areas than of the frontopolar region, and a relative integrity of the tangential fibers with a loss mainly in the meshwork even in the frontopolar area. One definitely expansive case shows more infiltration. Severe mental involvement seems to go with affection of the second and third layer ("the latter largely in its peripheral parts"). The case with frequent paralytic attacks seems to show most intensively patches of demyelinization.

The clinical data are so meager that an attempt at correlation is uncertain and hazardous. A perusal of the 138 pages of description is out of the question. It seems remarkable that such a detailed record should be rendered without a more adequate summing up. The illustrations are similarly profuse: first, forty-four pictures of the normal cortex in the areas used and twenty-three figures from the general paralysis material. For the sake of a more solid basis of comparison and a correct interpretation of the photos, a specific discussion of each case, the conditions of death with their specific influence in the pictures distinct from the lesions constituting the general paralytic process, and a summing up of the contrasts would have been infinitely more informing than the unsystematized mass of detail. One of the striking impressions of the pictures is the great improbability of a diagnosis of locality and process if submitted without text to even competent students of the human cortex. Their utilization would have been greatly facilitated by a summarizing statement of the essential facts and conclusions with each case.

DEMENTIA PRAECOX, INTERMEDIAERE PSYCHISCHE SCHICHT UND KLEINHIRN-BASALGANGLIEN-STIRNHIRNSYSTEME. By Dr. Max Loewy (Marienbad). Paper, pp. 120. Berlin: S. Karger, 1923.

We may well envy those who will be able to look back on the present-day discussions concerning dementia praecox with all the pretensions, presuppositions, and implications involved in these discussions. As one of the high points of the products along this line we might well consider the present monograph, as it makes capital of practically all the loose ends and gropings of psychology, neurology, and clinical speculation and generously touches the equally fertile territories of the doctrine of constitutions, of endocrinology and the Abderhalden ferment-doctrine, leaving very little for any possibly more comprehensive spirit.

The goal of Max Loewy is to point on the one hand to an "intermediary psychic layer" between Wernicke's somatopsyche and allopsyche and his autopsyche, and to find, on the other hand, a neurologizing equivalent in the cerebellar-striatal-frontal lobe system. Well read and with a very detailed range of familiarity with psychiatric observation, he indulges in a discussion which unfortunately, in contrast to the older studies of Kleist, does not work with casuistic evidence, but uses freely the host of facts which might play on each other in a mind with many rather loose and free associations, frequently of doubtful cogency. Many of the pages resemble notes collected in reading, with annotations but an inadequate effort to work for a solid basis which the reader and the writer might be sure to be able to share.

Loewy considers the assumption of an intermediary psychic stratum and its derangement an auxiliary hypothesis with which he may study the symptoms and pathogenesis and localization of dementia praecox together with Kleist's doctrine of the cerebellar-thalamo-striatal-frontal systems. He uses extensively the facts and hypotheses concerning paralysis agitans to supply fragments for a common interpretation, and he would then like to decide how the histopathology of dementia praecox might throw light on the possible existence of an elective system-disease and possibly an heredodegeneration with correlation of the disorders of the intermediary psychic stratum and a disease of "the basal ganglionic systems with their understructure and overstructure." Without a thorough study of Loewy's older contributions the present struggle for internal readjustment of his ideas and of the quotations from writers would be difficult to follow, and since no sufficient new factual basis is offered, the work had best be looked on as ponderings which unfortunately do not tend toward simplicity, but suffer from distractibility by too many loose collateral considerations which fail to change into a solid rock the sand on which the structure rests. An attempt to translate the summary shows at once the impossibility of solid common ground between reader and writer.

1. The symptoms of dementia praecox can be derived partly from the "psychomotor disorders" in the sense of Wernicke and Kleist, i. e., from disorders of the automatic mimic and reactive movements, etc., the "Einstellbewegungen" or thought movements and associative movements or empathic movements, the expressive and affective movements; partly from disorders of the coenesthesis and its course, among them possibly also disorders of labyrinthine origin; partly from disorders of affect formation and disposal of affects, in the form of impoverishment of affect, rigidity of affect, defective capacity of modulation of affect; partly from disorders of formation of ideas in the form of disorders of direction and selection—all this in that part of the mental processes which prepare the ground for conscious thinking and experience.

2. The disorders which underlie these symptoms are most variously interrelated. They are disorders in a common functional domain, the common intermediary stratum which furnishes the forerunner and foundation for the conscious thought processes as they diverge in various directions and constitute the unnoticed (unconscious) common understructure, the common basement ("cellar construction") of the variously directed and diverging (or differential) conscious life. This intermediate psychic stratum may be placed schematically between Wernicke's allopsyche (the external world) and somatopsyche (the consciousness of one's own body) on the one hand and Wernicke's autopsyche (the conscious personality) on the other. In this situation the intermediary functions approach Stransky's thymopsyche and that which we call character, but they are not quite identical, because they include the psychomotility, the coenesthesis, the first stages of affects and feelings and moods (Gemütslagen and Kronfeld's Gestimmtheiten) and the sympathetic (co-vibrating) thought atmospheres, the fringe of James, the appositional layer of condensed steam of nonattended to nature surrounding the conscious thinking, the mother-lye solution out of which the conscious thought is crystallized.

3. Disorders in the realm of functions of the intermediate psychic stratum are occasionally found also in diseases outside of dementia praecox, especially instructively for the symptomatology of dementia praecox in the diseases of the basal ganglions.

4. Kleist sees in the psychomotor disorders generally and in dementia praecox a damage of the cerebello-basal-frontal systems. This doctrine finds, according to Loewy's conceptions, a psychologic correlate, through the interpretation of the symptoms of dementia praecox as disorders in the realm of function and effective influence of an intermediary psychic stratum. The assumption of disorders in the functional domain of the intermediary psychic stratum and the basal systems (including, of course, also their spheres of influence in the cortex) in dementia praecox appears to lend itself to give a unitary principle or explanation to the polymorphous symptoms, temporary pictures, types of course and outcome, and the pathogenetic interpretations (through various "primary symptoms"): through differences in the distribution, the direction of distribution and progression, the intensity, the acuteness or chronicity of the damages of the sphere of function of the intermediary psychic stratum and of the cerebello-basal-frontal system. From the interlacing of the intermediary functions we can also understand that the same symptom in various patients may arise from different functional disorders of the intermediary stratum.

5. There also evolves from this the relation of the schizoid temperaments (the schizothymic of Kretschmer, the schizoid reaction-types of Eugen Kahn, the schizophrenic reaction-types of Popper, the heredoschizosis and phenoschizosis of Bleuler) to the manifest schizophrenia—in short, the prepsychotic personality and its relatives who do not become overtly diseased, with the manifest dementia praecox on the ground of a possibly hypothesized inherited weakness and susceptibility of certain brain systems, especially the cerebellum-basal ganglion-frontal lobe systems of Kleist.

Partly through the normal vital stimuli, the normal course of life, an elective system-disease might arise, with pronounced abiotrophy (in the sense of Gowers) as exhaustion-disease (Aufbrauchkrankheit of Edinger); partly through disease and damage of a susceptible system capable of little resistance, in the sense of a locus minoris resistentiae after the manner of other nervous system diseases following injury, infections, or the physiologic revolutions of

puberty, gravidity, puerperal state, climacteric and perhaps also senility, or otherwise endocrine or autotoxic, in short exogenous and endogenous through damaging influences and disorders of functions of an elective kind.

6. It is not excluded (ausgeschlossen) that the endocrine disorders which have been ascertained in dementia praecox might be secondarily produced by participation of the interbrain. It may be possible however that more (among other things possibly also the periodicity of many types of course) speaks in favor of the causal significance of the endocrine disorders, which would then deserve the attribute of an elective affinity especially for the domain of function of the intermediary psychic stratum and the cerebellum-basal ganglion-frontal brain systems. The nature of these pluriglandular endocrine disorders with affinity for the just mentioned systems must remain "open," just as the causal significance of the internal secretions for dementia praecox, etc., etc. More than four additional pages of similar plausibilities with tributes to Kleist, Freud, Kraepelin, Wernicke, Bleuler, Berze, Kronfeld, et al., summarize the suggestions and precautions of this flight of hypothetical rumination.

It may be that certain minds will profit from this kind of stimulation, and the reviewer does not want to run down these confessions of a friend of speculation. Yet in these days there seems to be more need of concreteness and direct attack on demonstrable facts, and it is well that the reader should not be misled to expect a calm and sober demonstration of facts such as the title might make one long for and expect. On the other hand, one finds evidence of extensive reading, many bright references, but also little idea of what an American editor would say of the advisability of incurring the expense of printing these 120 pages. The European writer still enjoys privileges that would not be easily obtained in this country.

LA SUBCONCIENCIA. Discurso de recepción del Dr. D. Rafael del Valle y Aldabalde y contestación del Dr. D. Ricardo Pérez-Valdés y Aguirre, leídos el 18 Febrero de 1923. Publication of the Real Academia Nacional de Medicina, Madrid, Establecimiento tipográfico nieto y compañia, 1923.

As an oration on his admission to the National Royal Academy of Medicine of Spain, the author gives a rather formal review of subconsciousness. The author of a "Psychotherapy for the Practitioner" (1920), Valle y Aldabalde shows wide reading in American, French, and German literature. He states in conclusion that "the human soul as the substantial form of the body intervenes or enters into all the phenomena and processes of the organisms, not as force but as direction. Its entering or intervention is more obvious in the phenomena called psychic, and among these it is much more so in those called conscious. The subconscious comes to be the point of communication or connecting link between the conscious and the somatic or organic, a resonator for the emotions and sentiments, a source of psychic energy for the instincts and passions, a wealth of images, of ideas and of associations of the ones and the others which lie latent in its depth ready at any moment to be utilized for the conscious 'psychism' and mechanism which through the medium of habit saves the superior psychism a great share of its work.

"Thanks to the subconscious, the human understanding, served and directed by the rational and free will, does not have to interfere not only in the phenomena of organic life, but as little in the very numerous acts of the properly or really psychic life, so that in that way it can devote itself to the noble and lofty tasks which place it in a position quite apart and superior, not in degree,

but in its nature, to that which the highest beings of creation occupy; thus reproducing, although in a slight degree, in harmony with the limitation and finiteness of our power and understanding, the relation which exists between the secondary causes which govern the universe and world, with all its marvelous machinery, its grandiose harmony, and its immenseness, of which we get but a faint idea through the thousands of years of light which cover the distance between us and some stars, and the sovereign intelligence of the Supreme Creator who created it with his sublime 'fiat' and who conserves it without the need of a new act of his omnipotent power nor of a new manifestation of his infinite intelligence."

This is the epilogue to the discussion of the following topics: Previous psychologic and anatomophysiologic notions; organic sensibility and special senses, affective sensibility and emotions, memory, imagination, instincts, passions, intelligence and rational volition, habit, age and sex, temperaments, sleep and dreams, hypnotism, suggestion, psychanalysis, hysterisms, double personality, obsessions, hallucinations and illusions, delirium and mental diseases. There is a remarkably wide range of information carried together in a fluent oration, reaching as far into the physiologic and endocrinologic foundations as into the loftiest connections.

The response by Dr. Pérez-Valdés y Aguirre gives a very appreciative account of Valle's life and work as editor, translator of foreign medical workers, and leading member of the profession, and as a fervent Christian and Catholic—a position similar to that of our Dr. James J. Walsh. Pérez-Valdés then proceeds to cap the climax of the exaltation of the subconscious to the position of being "the real psychology itself," by giving an account of how apparently the observations of Janet and Ribot and a peculiar case of delusion, of being dead, but with fear of being buried alive, lead over to the recent developments of metapsychics, with evidently profound respect for the latest developments of ectoplasm, experiences of prescience quoted by Richet, etc.

Among the historical references of Valle there is an interesting quotation from Avicena's Canon, giving an account of the use of irregularity of the pulse for the diagnosis of the mental disease of being in love, when the names were mentioned that might touch the emotions—given as an instance of eleventh century psychanalysis among Arabs.

As a picture of the catholicity of certain exponents of the subconscious the pamphlet is of decided interest. It also is an illustration of the quickness and expeditiousness of publications of proceedings of a society.

INDEX TO VOLUME 10

The star (*) preceding the page number indicates an original article in THE ARCHIVES. Book Reviews, Obituaries and Society Transactions are indexed under these headings in their alphabetical order under the letters B. O and S, respectively.

PAGE

Abreaction, phenomenon of; Gordon 108
Achondroplasia in mother and child; Bassoe 114
Actinomycosis of cerebrum, case of; Klemmer 479
Address; Zabriskie 129
Alcoholism and behavior of white rats; maze-behavior of treated rats and their offspring; MacDowell 705
Amaurotic idiocy, Tay-Sachs disease; study of symptomatology and pathology of 3 atypical cases; Torrance 698
Amebiasis, chronic intestinal, clinical aspects with special reference to neuropsychiatric manifestations; H. W. Wright *226
Anaphylaxis, neuropathic manifestations in infants and children as result of anaphylactic reaction to foods contained in their dietary; Shannon 595
Anatomy, comparative, anatomical studies on motor cortex of macacus rhesus; Nañagas 108
comparative, certain features of laterosensory canals of Plagiostomi; Allis 107
comparative, morphogenesis of nervous centers in chiroptera; first phases of development of nervous centers in rhinolophus hipposideros (Bechstein); Luna 689
comparative, note on excitable areas of cerebral hemispheres of pigeon; Rogers 112
comparative, origin and distribution of tractus solitarius in guinea-pig; Allen 111
Angiospasm in vasomotor and trophic neuroses; Brüning 237
Animal mechanism; Sherrington 252
Appendicitis, acute, skin signs of viscerosensory phenomena in; Livingston 702
Apraxia; Papadato 474
clinical and anatomic studies of; Brun 344
Association for Research in Nervous and Mental Diseases 685
Asthma and vago-sympathetic imbalance; Galup 696
Atrophy, hereditary muscular, familial myopathy of peroneal or distal type; Rimbaud and Giraud 470
Ayer, J. B.: Spinal subarachnoid block, its significance as a diagnostic sign *420
—and Cushing, H.: Xanthochromia and increased protein in spinal fluid above tumors of cauda equina *167

Barkan, O., and Mehrtens, H. G.: Researches on pupillary reactions in epidemic encephalitis *399
Bennett, A. E.: Neurosyphilis in aged, study of 19 cases of senile neurosyphilis occurring in series of 2,175 cases of all types of nervous syphilis *324
Beverly, B. I., and Sherman, M.: Factor of deterioration in children showing behavior difficulties after epidemic encephalitis *329
Blood chemistry in epilepsy; Lennox 264
BOOK REVIEWS:
Dementia Praecox, intermediäre psychische Schicht und Kleinhirn-Basalganglien-Stirnhirnsysteme; M. Loewy 721
Der Selbstmord; R. Weichbrodt 379
Die Hirnkarte des Paralytikers. Studien über das Wesen und die Ausbreitung des paralytischen Prozesses in der Hirnrinde; S. Saito .. 719
Diseases of Nervous System; Text Book of Neurology and Psychiatry; S. E. Jelliffe and W. A. White 603

Book Reviews—Continued PAGE
Einführung in die Klinik der inneren Sekretion; G. Peritz..... 379
Form and Functions of Central Nervous System; Introduction to Study of Nervous Diseases; F. Tilney et al..... 488
La Subconciencia; D. R. del Valle y Aldabalde and D. R. Pérez-Valdés y Aguirre 723
Oppenheim's Lehrbuch der Nervenkrankheiten für Aerzte und Studierende, Vol. 1; R. Cassirer et al..... 489
Brain abscess, treatment by induction of protective adhesions between brain cortex and dura before establishment of drainage; Dowman.....*697
centers, crural monoplegia and paraplegia of cortical origin with discussion of cortical centers for rectum, bladder and sexual functions; G. Wilson *669
centers, diencephalic centers controlling associated locomotor movements, an experimental research; H. Mella.....*141
cortex, note on excitable areas of cerebral hemispheres of pigeon; Rogers 112
cortex, of macacus rhesus, anatomical studies on motor cortex; Nañagas 108
lesions, spontaneous cerebral lesions in monkeys, their significance in experimental pathology; B. Lucke.....*212
lesions, two specimens of occipital lobe lesions; Favill..... 115
significance of frontal lobes for higher psychic functions; Donath..... 587
tumor and serous meningitis; Claude and Schoeffer..... 592
tumor, notes on series of intra-cranial tumors and conditions simulating them; tumor suspects: tumors unverified: tumors verified; H. Cushing *605
tumor, radiographic diagnosis of site and nature of variety of cerebral tumor (psammomian or angiolithic sarcoma); Souques..... 374
tumor, simulating dementia paralytica, with operation and complete neurologic and mental recovery; Rosenheck..... 111
tumor, value of ventriculography, clinical experience based on a series of 40 cases; F. C. Grant.....*154
tumor, venous angioma of cerebral cortex; Drought and Ballance..... 594
tumor, visual hallucinations as cerebral localizing phenomenon, with especial reference to their occurrence in tumors of temporal lobes; G. Horrax *532
tumors in young children; clinical and pathological study; Wollstein and Bartlett 588
tumors, specimens of, removed at operation; Oglesby..... 483
Brown-Sequard syndrome, remarks on recovery of case; Marie, Bouttier and Mathieu 367

Cauda equina, tumors, xanthochromia and increased protein in spinal fluid above tumors of cauda equina; H. Cushing and J. B. Ayer.....*167
Central Neuropsychiatric Association, second annual convention, St. Louis, October, 20, 1923..... 687
Centrifugation of nervous system—method for neurocytologic study, an experimental investigation of cellular changes in commotion; S. Ingvar *267
Cerebellum, effects of ablations of cerebellar cortex after suppression of circulation; Rossi 373
otitic abscess of; Yerger..... 107
Cerebrospinal fluid, acetic anhydride-sulphuric acid test; Boltz..... 594
alterations in currents and absorption of cerebrospinal fluid following salt administration; Foley 251
arsenic content after intravenous administration of silver arsphenamine; (clinical and biochemical study of neurosyphilis); Cornwall and Meyers 707
colloidal gold reaction in acute poliomyelitis; Regan, Litvak and Regan.. 251
of encephalitis lethargica; Ayer and Cockrill..... 254
pressure in experimental epilepsy, some preliminary observations on cerebrospinal fluid, venous and arterial pressure in experimental convulsions; Macdonald 262
spinal subarachnoid block, its significance as a diagnostic sign, analysis of 53 cases; J. B. Ayer.....*420
spirochetosis of; Riddel and Stewart..... 111

PAGE

Cerebrospinal fluid—Continued
Wassermann reaction and colloidal methods in spinal fluid of neurosyphilitics; Rizzo 467
xanthochromia and increased protein in spinal fluid above tumors of cauda equina; H. Cushing and J. B. Ayer *167
xanthochromia, its origin and significance; F. J. Scully *83
Children, dysthymic; de Sanctis 475
mental disorders of; Glueck 471
Chiroptera, morphogenesis of nervous centers in chiroptera; first phases of development of nervous centers in rhinolophus hipposideros (Bechstein); Luna 689
Chorea cruciata; Lhermitte and Bourguina 596
Huntington's, and migraine; Benedek and Goldenberg 591
Convulsions, early convulsions in epileptics and in others; Patrick and Levy 715
Corpus striatum, studies of brain stem; experimental study of corpus striatum of pigeon as related to various instinctive types of behavior; Rogers 105
diseases of striate system; Vogt and Vogt 563
syndrome, studies in extra-pyramidal syndromes; progressive infantile torsion spasm; Wimmer 235
Cranium, intracranial tumors, notes on series of intracranial tumors and conditions simulating them; tumor suspects: tumors unverified: tumors verified; H. Cushing *605
hyperostoses overlying endothelioma of dura; Phemister 372
osteoma; Rand 377
Crural monoplegia and paraplegia of cortical origin; G. Wilson *669
Cushing, H.: Notes on series of intracranial tumors and conditions simulating them; tumor suspects: tumors unverified: tumors verified. *605
Neurological surgeons *381
—and Ayer, J. B.: Xanthochromia and increased protein in spinal fluid above tumors of cauda equina *167

Dana, C. L.: Dr. George M. Beard, a sketch of his life and character with some personal reminiscences *427
Davis, L. E., and Pollock, L. J.: Studies in decerebration; a method of decerebration *391
Decerebration, studies in; a method of decerebration; L. J. Pollock and L. E. Davis *391
decerebrate rigidity of Sherrington in man, its recognition and differentiation from other forms of tonic muscular contraction; F. M. R. Walshe *1
Delinquency, factor of deterioration in children showing behavior difficulties after epidemic encephalitis; M. Sherman and B. I. Beverly .. *329
Dementia paralytica simulated by cerebral neoplasm, with operation and complete neurologic and mental recovery; Rosenheck 111
praecox, catatonic; physiotherapeutics, and results obtained in a series of 20 cases; Main 113
praecox, mental health of 463 children from dementia-praecox stock; Canavan and Clark 112
praecox, studies of schizophrenic reactions; Hall and Neyman 248
praecox, study in basal metabolism in, and manic-depressive psychosis; Gibbs and Lemcke 252
traumatic, case of; Benon and LeFévre 242
Dowman, C. E.: Complete transverse lesion of spinal cord with retention of superficial reflexes *33
Dura, endothelioma, nature of cranial hyperostoses overlying; Phemister .. 372
subdural effusion and hydrocephalus; presentation of a recovered case; Penfield 128
Dystonia, case with athetoid features; Margaretten 709
Dystrophy, muscular, case showing evidence of, and pituitary disturbance; Patten 485

Embryology, brain stem and cerebellum of 5 months' fetus; Freeman 123
subdivisions of neural folds in man; Bartelmez 593
Encephalitis, acute hemorrhagic; Toomey, Dembo and McConnell 472
acute, three cases of acute encephalitis treated with specific serum; Helmholz and Rosenow 378
acute, unusual case of; clinico-pathologic report; Winkelman 486

Encephalitis—Continued PAGE
epidemic, a case exhibiting a conduct disorder; Leavitt..... 122
epidemic, and herpes febrilis; Szymanowski and Zylberlast-Zand..... 590
epidemic, case of, with tremor typical of multiple sclerosis; Hassin and Stone 369
epidemic, clinical features of; Lloyd..... 124
epidemic, contribution to neuropathology of parkinsonian syndrome following; Strauss and Globus..... 597
epidemic, experiments in treatment with casein intraspinally; Roch.... 593
epidemic, factor of deterioration in children showing behavior difficulties after epidemic encephalitis; M. Sherman and B. I. Beverly.....*329
epidemic, high grade choked disks in; Spiller..... 124, 706
epidemic, in children; neuropsychiatric sequelae of; Ebaugh..... 465
epidemic, meningeal form treated with antimeningococcic serum; Herrick 371
epidemic, researches on pupillary reactions in; H. G. Mehrtens and O. Barkan *399
epidemic, simulated by rheumatic fever with cerebral symptoms; Frissel 706
epidemic, spinal fluid of; Ayer and Cockrill..... 254
lethargic: See Encephalitis, epidemic
tuberculous, case of porencephalic defect associated with; histopathologic support to Strümpell theory of inflammation; R. P. Winterode and N. D. C. Lewis.....*304
Encephalomyelitis, fulminating; H. E. Meleney.....*411
Endocrine disturbances and late epilepsy; Perrin and Richard..... 245
imbalance and mental disease, a correlative study; Lewis and Davies.. 463
Epilepsy and gunshot wounds of head; Turner..... 104
case of 22 years' standing due to calcified endothelioma or perithelioma in left lateral ventricle, removal and recovery; Lynn-Thomas..... 592
diagnosis of; Patrick and Levy..... 371
diets and fasting in..... 686
early convulsions in epileptics and in others; Patrick and Levy..... 715
experimental, cerebrospinal fluid pressure in, some preliminary observations on cerebrospinal fluid, venous and arterial pressure in experimental convulsions; Macdonald..... 262
late, and endocrine disturbances; Perrin and Richard..... 245
psychogenic, some unusual forms of; Clark..... 712
studies in chemistry of blood in; Lennox..... 264
Ether, action of, on circulation in traumatic shock; Cattell..... 460
Exhaustion, studies in; hemorrhage; Crile..... 704
studies in; physical trauma; Crile..... 249

Farr, C. B., and Lueders, C. W.: Gastric secretory functions in psychoses.*548
Fasciculus solitarius, origin and distribution of tractus solitarius in guinea-pig; Allen 111
Feeblemindedness: See also Mongolism
syphilis and mental deficiency; Key and Pijper..... 706
types of, in children; Gött..... 240
Folie à trois; F. J. Gerty and G. W. Hall.....*436
Frazier, C. H., and Spiller, W. G.: Telangiectasis of spinal cord..... *29
Free, G. B. M., Pike, H. V., and Jackson, J. A.: Psychic manifestations in paralysis agitans *680
Fröhlich's syndrome: See under Pituitary body

Gerty, F. J., and Hall, G. W.: Folie à trois.....*436
Gilman, P. K., and Schaller, W. F.: Spastic paraplegia in flexion; case due to meningeal tumor compressing lower cervical cord on anterior and right lateral aspects *512
Goiter, exophthalmic, studies of exophthalmic goiter and involuntary nervous system; Kessel, Lieb and Hyman..... 703
exophthalmic, rôle of labyrinth in; Hellin and Szware..... 473
exophthalmic, study of 50 consecutive cases of exophthalmic goiter; Kessel, Hyman and Lande..... 703
toxic, further observations on roentgen-ray treatment of toxic goiter; Means and Holmes 585
Grant, F. C.: Value of ventriculography, clinical experience based on a series of 40 cases.....*154

PAGE

Hall, G. W., and Gerty, F. J.: Folie à trois.....*436
Hallucinations, visual, as cerebral localizing phenomenon, with especial reference to their occurrence in tumors of temporal lobes; G. Horrax.*532
Hassin, G. B.: Concussion of spinal cord, a case with clinical picture of amyotrophic lateral sclerosis *194
Head wounds, remote effects of gunshot wounds of head; Rawlings..... 701
positions of, and muscle tone; Simons..... 358
Headache, lower half headache (neuralgic) of nasal origin; glossodynia, otalgia, nausea, parageusia, vertigo, scotoma, photophobia, rhinorrhea and asthma as isolated related phenomena; Sluder..... 476
Hemihypertrophy and hemihypertonia, case of, developing after typhoid fever; Rea 477
Hemiparaplegia: See Brown-Sequard syndrome
Hemorrhage, studies in exhaustion; hemorrhage; Crile..... 704
Herpes febrilis and lethargic encephalitis; Szymanowski and Zylberlast-Zand 590
zoster; Ramond 472
Hippocampus, significance of the term; Lewis..... 474
Horrax, G.: Visual hallucinations as cerebral localizing phenomenon, with especial reference to their occurrence in tumors of temporal lobes....*532
Hunt, J. R.: Dual nature of efferent nervous system, further study of static and kinetic systems, their function and symptomatology contents.... *37
Hydrocephalus, ventriculoscopy and intraventricular photography in internal hydrocephalus; Fay and Grant..... 113
subdural effusion and hydrocephalus; presentation of a recovered case; Penfield 128
Hypochondria, significance and management of hypochondriacal trends in children; Richards 103
Hypophysis: See Pituitary Body
Hyslop, G. H., and Kraus, W. M.: Pathology of motor paralysis by lead..*444

Idiocy: See Amaurotic idiocy; Feeblemindedness; Mongolism
Index finger, phenomenon of; Raimiste..... 588
Infundibulum, syndromes of infundibular-hypophysial group in epidemic encephalitis; Mouzon 475
Ingvar, S.: Centrifugation of nervous system—method for neurocytologic study, an experimental investigation of cellular changes in commotion *267
Injections, regions of cerebrospinal system in which therapeutic injections should be done; Solomon..... 249
Insanity, folie à trois; F. J. Gerty and G. W. Hall.....*436
manic-depressive, (acute) cured by thyroidectomy; Stocker..... 248
manic-depressive, study in basal metabolism in dementia praecox and manic-depressive psychosis; Gibbs and Lemcke..... 252
Instinctive reactions, so-called, some chief characteristics of; Paton..... 599
Intestinal parasites, chronic intestinal amebiasis, clinical aspects with a special reference to neuropsychiatric manifestations; H. W. Wright.*226

Jackson, J. A., Free, G. B. M., and Pike, H. V.: Psychic manifestations in paralysis agitans *680

Kolmer, J. A., and Lucke, B.: Histologic changes produced experimentally in central nervous system of monkeys by mercury.....*288
Kraus, W. M., and Hyslop, G. H.: Pathology of motor paralysis by lead..*444

Labyrinth, rôle of, in exophthalmic goiter; Hellin and Szware..... 473
Lead, pathology of motor paralysis by lead; G. H. Hyslop and W. M. Kraus.*444
Lewis, N. D. C., and Winterode, R. P.: Porencephalic defect associated with tuberculous encephalitis, histopathologic support to Strümpell theory of inflammation *304
Locomotion, diencephalic centers controlling associated locomotor movements, an experimental research; H. Mella.....*141
Lucke, B.: Spontaneous cerebral lesions in monkeys, their significance in experimental pathology *212
—and Kolmer, J. A.: Histologic changes produced experimentally in central nervous system of monkeys by mercury.....*288

PAGE

Lueders, C. W., and Farr, C. B.: Gastric secretory functions in psychoses. *548
Lung, ganglia, plexuses, and nerve-terminations of mammalian lung and pleura pulmonalis; Larsell 103

Magnesium sulphate as a sedative; Weston and Howard 377
Meckel's ganglion, lower half headache (neuralgic) of nasal origin; glossodynia, otalgia, nausea, parageusia, vertigo, scotoma, photophobia, rhinorrhea and asthma as isolated related phenomena; Sluder 476
Mehrtens, H. G., and Barkan, O.: Researches on pupillary reactions in epidemic encephalitis *399
Meleney, H. E.: Fulminating encephalomyelitis *411
Mella, H.: Diencephalic centers controlling associated locomotor movements *141
Meninges, calcification and ossification of; Halstead and Christopher 707
Meningitis in early infancy, incidence of, with description of 2 cases due to unusual organisms; Cooke and Bell 247
influenzal; Rivers 591
meningococcus, intraventricular and subdural serum treatment of, in infancy; Howell and Cohen 247
meningococcus, introduction of antimeningococcus serum by cistern puncture; report of case in infant aged 4 months cured by this method; Mitchell and Reilly 596
serous, and brain tumor; Claude and Schoeffer 592
Mental hygiene, organization and scope of State Bureau of Mental Health; Sandy 109
significance and management of hypochondriacal trends in children; Richards 103
work of demonstration clinics conducted by Division on Prevention of Delinquency of National Committee for Mental Hygiene; Anderson 133
Mercury, histologic changes produced experimentally in central nervous system of monkeys by mercury; B. Lucke and J. A. Kolmer *288
Metabolism, basal, in dementia præcox and manic-depressive psychosis; Gibbs and Lemcke 252
Migraine, abdominal crises of; Buchanan 374
and Huntington's chorea; Benedek and Goldenberg 591
blindness in; Löhlein 243
in children; Curschmann 458
treatment with peptone; evidence of anaphylactic character of seizure; Miller and Raulston 707
Mongolism in one of twins and etiology of mongolism; Halbertsma 708
sella turcica of mongolian imbeciles; G. S. Stevenson and A. D. Stultz *299
Monkeys, spontaneous cerebral lesions in monkeys, their significance in experimental pathology; B. Lucke *212
Monoplegia, crural monoplegia and paraplegia of cortical origin with discussion of cortical centers for rectum, bladder and sexual functions; G. Wilson *669
Muscles, artificial nerve branches for innervation of paralyzed muscles; Stookey 700
electromyographic studies of muscular fatigue in man; Cobb and Forbes 264
tone and positions of head; Simons 358
tonic contraction, decerebrate rigidity of Sherrington in man, its recognition and differentiation; F. M. R. Walshe *1
Myasthenia gravis, observations on creatinuria and glycosuria in myasthenia gravis; Williams and Dyke 111
gravis, two juvenile cases of; Neustaedter 709
Myelin sheath, fine structure of medullary sheaths of nerve fibers in normal and pathologic conditions, and a simple staining method for; Kamijo 363
Myelitis: See Spinal Cord
Myotonia, atrophica (dystrophia myotonica); Adie and Greenfield 586

Nerve cell, centrifugation of nervous system—method for neurocytologic study, an experimental investigation of cellular changes in commotion; S. Ingvar *267
cicatrization, anatomic and functional results observed in course of cicatrization of nerves in dog; Guyon 239

Nerve—Continued PAGE
fiber, fine structure of medullary sheaths of, in normal and pathologic conditions, and a simple staining method for myelin sheaths; Kamijo 363
intercostal nerve variations; H. J. Prentiss *232
median, pattern of weakness of hand in ulnar and median nerve lesions; Pollock 121
peripheral, causalgia and allied painful conditions due to lesions of; Carter 106
trigeminus, in albino rat, bilateral effects from unilateral section of branches of; Nittono 252
ulnar, pattern of weakness of hand in ulnar and median nerve lesions; Pollock 121
Nervous system, efferent, dual nature of efferent nervous system, further study of static and kinetic systems, their function and symptomatology contents; J. R. Hunt *37
syphilis, familial, from various extra familial sources, clinical contribution to question of neurotropism; Moore and Keidel 373
syphilis, in aged, study of 19 cases of senile neurosyphilis occurring in series of 2,175 cases of all types of nervous syphilis; A. E. Bennett *324
syphilis, intraspinal therapy in; Fordyce 112
syphilis, pontobulbar crises associated with sialorrhea in; Boas and Kraus 109
syphilis, Sachs-Georgi reaction in; Levinson and Petersen 376
syphilis, tryparsamid in; Lorenz, Loevenhardt, Bleckwenn and Hodges 702
syphilis, types of, in China; Woods 253
syphilis, Wassermann reaction and colloidal method in spinal fluid of neurosyphilitics, theoretic considerations on Wassermann test; Rizzo 467
tumors, experimental studies on malignant tumors of central nervous system; Flatau 456
Neuralgia, lower half headache (neuralgic) of nasal origin; glossodynia, otalgia, nausea, parageusia, vertigo, scotoma, photophobia, rhinorrhea and asthma as isolated related phenomena; Sluder 476
Neurological surgeons: with report of one case; H. Cushing *381
Neuroses, angiospasm in vasomotor and trophic neuroses; Brüning 237
Neurotropism, studies in familial neurosyphilis, familial neurosyphilis from various extrafamilial sources, clinical contribution to question of neurotropism; Moore and Keidel 373

Obituaries:
Dr. George M. Beard, a sketch of his life and character, with some personal reminiscences; C. L. Dana *427
Heinrich Obersteiner, 1847-1922 100
Paul Waterman, M. D., 1877-1923 714
Olive, superior, cell structure of, in man; Malone 596

Pachymeningitis, internal hemorrhagic, in infancy; Burhans and Gerstenberger 375
Pain, causalgia and allied painful conditions due to lesions of peripheral nerves; Carter 106
central, from lesions of pons; T. H. Weisenburg and S. S. Stack *500
central, in syringomyelia and dysesthesia and overreaction to sensory stimuli in lesions below optic thalamus; W. G. Spiller *491
Palilalia, contribution to knowledge of; Senise 375
Paralysis agitans, psychic manifestations in paralysis agitans; J. A. Jackson, G. B. M. Free and H. V. Pike *680
general; Riser and Gay 246
general, case having an evolution of 17 years; Cornil and Robin 595
lead, pathology of motor paralysis by lead; G. H. Hyslop and W. M. Kraus *444
postdiphtheric, characteristics of cerebrospinal fluid in; Regan, Regan and Wilson 594
Paraplegia, clinical study of case due to bullet and coming on 4 years after wound; Barré 699
crural monoplegia and paraplegia of cortical origin with discussion of cortical centers for rectum, bladder and sexual functions; G. Wilson. *669
spastic, in flexion; case due to meningeal tumor compressing lower cervical cord on anterior and right lateral aspects; W. F. Schaller and P. K. Gilman *512

PAGE

Parkinson's "Shaking Palsy"..... 562
Parotid tumors, preservation of facial nerve in radical treatment of; Adson and Ott..... 708
Pike, H. V., Jackson, J. A., and Free, G. B. M.: Psychic manifestations in paralysis agitans.....*680
Pilomotor centers and pilomotor pathways; Thomas..... 252
Pituitary body, integrity of, with tumor of infundibulum (tuber cinereum) and adipose syndrome; Ley..... 462
coagulatory action of pituitary extract; Hanns, Stéfanovitch and Arnovljevitch..... 703
syndromes of infundibular-hypophysial group in epidemic encephalitis; Mouzon..... 475
tumors and psychic disturbances; Sainton and Péron..... 589
Plagiostoma, certain features of laterosensory canals of Plagiostomi; Allis..... 107
Pleura, ganglia, plexuses, and nerve-terminations of; Larsell..... 103
Poliomyelitis, acute, colloidal gold reaction in; Regan, Litvak and Regan 251
Pollock, L. J., and Davis, L. E.: Studies in decerebration; method of decerebration.....*391
Pons, central pain from lesions of; T. H. Weisenburg and S. S. Stack....*500
pontobulbar crises associated with sialorrhea in syphilis of nervous system; Boas and Kraus..... 109
Prentiss, H. J.: Intercostal nerve variations.....*232
Psychiatry, follow-up work in mental and surgical cases; Bond..... 106
Psychoneuroses, neuropsychiatric analysis of 2,500 ex-service men with special study of relation of thyroid to psychoneuroses; Truitt..... 118
Psychopathic inferior, constitutional; problem in diagnosis; Johnson..... 110
Psychoses, gastric secretory functions in; C. B. Farr and C. W. Lueders..*548
war and acute delirious hyperthymia; Benon..... 466
Pupil, dilator pupillae and sympathetic; Ingalls..... 110
researches on pupillary reactions in epidemic encephalitis; H. G. Mehrtens and O. Barkan.....*399

Raynaud's disease, phenomena of; Buchanan..... 590
Reflexes of spinal frogs as induced by chemical stimulation; Foley..... 253

Sachs-Georgi reaction in neurosyphilis; Levinson and Petersen..... 376
Sarcoma, psammomian, radiographic diagnosis of site and nature of variety of cerebral tumor (psammomian or angiolithic sarcoma); Souques.. 374
Schaller, W. F., and Gilman, P. K.: Spastic paraplegia in flexion; case due to meningeal tumor compressing lower cervical cord on anterior and right lateral aspects.....*512
Schizophrenia: See Dementia praecox
Sciatica, etiology and pathology of sciatica; Petren and Otterström..... 583
Sclerosis, amyotrophic lateral, clinical picture of, in case of concussion of spinal cord; G. B. Hassin.....*194
multiple, and its etiology; Long..... 233
multiple, case of epidemic encephalitis, with tremor typical of; Hassin and Stone..... 369
Scully, F. J.: Yellow spinal fluid, its origin and significance..... *83
Sella turcica of mongolian imbeciles; G. S. Stevenson and A. D. Stultz....*299
Sherman, M., and Beverly, B. I.: Factor of deterioration in children showing behavior difficulties after epidemic encephalitis.....*329
Shock, experimental traumatic shock; influence of morphin on blood pressure and alkali reserve; Cattell..... 705
experimental traumatic shock; action of ether on circulation in traumatic shock; Cattell..... 460
Silver arsphenamin, arsenic content of spinal fluid after intravenous administration of, (clinical and biochemical study of neurosyphilis); Cornwall and Meyers..... 707
Skin signs of viscerosensory phenomena in acute appendicitis; Livingston 702
Society Transactions:
American Neurological Association..... 597
Boston Society of Psychiatry and Neurology..... 254, 259
Chicago Neurological Society..... 114, 715

Society Transactions—Continued PAGE
New York Neurological Society..... 128, 709
Philadelphia Neurological Society..... 122, 477
Soldiers, neuropsychiatric analysis of 2,500 ex-service men with special study of relation of thyroid to psychoneuroses; Truitt..... 118
Spiller, W. G.: Central pain in syringomyelia and dysesthesia and over-reaction to sensory stimuli in lesions below optic thalamus.....*491
—and Frazier, C. H.: Telangiectasis of spinal cord..... *29
Spinal cord, complete transverse lesion of spinal cord with retention of superficial reflexes; C. E. Dowman..... *33
compression, second case of ascending compression myelitis associated with unusual pathology; Gosline and Murphy..... 257
compression, spastic paraplegia in flexion; case due to meningeal tumor compressing lower cervical cord on anterior and right lateral aspects; W. F. Schaller and P. K. Gilman.....*512
concussion of, a case with the clinical picture of amyotrophic lateral sclerosis; G. B. Hassin.....*194
in amblystoma, experiments on transplantation of, and their bearing upon stimuli involved in differentiation of nerve cells; Detwiler..... 469
spinal subarachnoid block, its significance as a diagnostic sign, analysis of 53 cases; J. B. Ayer.....*420
syndrome of subacute degeneration of long fibers of lateral motor bundles; Gordon 250
telangiectasis of; W. G. Spiller and C. H. Frazier..... *29
tumors, study of bladder and rectal disturbances in; B. Stookey.....*519
Stack, S. S., and Weisenburg, T. H.: Central pain from lesions of pons..*500
Stevenson, G. S., and Stultz, A. D.: Sella turcica of mongolian imbeciles.*299
Stomach, gastric secretory functions in psychoses; C. B. Farr and C. W. Lueders *548
Stookey, B.: Study of bladder and rectal disturbances in spinal cord tumors *519
Stultz, A. D., and Stevenson, G. S.: Sella turcica of mongolian imbeciles..*299
Suprarenal gland, growth of cortical and medullary substances and their volumetric relationship; Castaldi..... 370
Sympathectomy, periarterial; Halstead and Christopher..... 377
Sympathetic nervous system, case of lesion probably involving thalamus and subthalamic region with symptoms of irritation of sympathetic nervous system; Potts..... 477
Syphilis and mental deficiency; Key and Pijper..... 706
of the nervous system: See Nervous system syphilis
Syringomyelia, central pain in syringomyelia and dysesthesia and over-reaction to sensory stimuli in lesions below optic thalamus; W. G. Spiller *491
trophic changes in; Kooy..... 595

Thalamus, case of lesion probably involving thalamus and subthalamic region with symptoms of irritation of sympathetic nervous system; Potts 477
Thymus, enlarged, incidence of, without symptoms in infants and children; Greenthal 244
Thyroid gland: See also Goiter
acute manic-depressive insanity cured by thyroidectomy; Stocker..... 248
disease, observations of cardiovascular system in; Kerr and Hensel.... 705
weight variability of normal thyroid gland and its significance; Castaldi 368
Torsion spasm, studies in extrapyramidal syndromes; progressive infantile torsion spasm (syndrome of corpus striatum); Wimmer..... 235
Trauma, studies in exhaustion; physical trauma; Crile..... 249
Tremor, experimental vestibular, with analogous clinical cases; C. L. Woolsey *314
fibrillary, do fibrillary contractions always denote organic change in nervous system? Ostheimer..... 480
Tryparsamid in neurosyphilis; Lorenz, Loevenhardt, Bleckwenn and Hodges 702
Tumor: See Brain tumor; Nervous system tumor, etc.
Typhoid fever, case of hemihypertrophy and hemihypertonia developing after; Rea 477

PAGE

Ventriculography, value of, clinical experience based on a series of 40 cases; F. C. Grant *154
Vertebra, cancer, radiologic signs of vertebral cancer; Sicard, Lermoyez and Laplane 468
Vestibular apparatus, quadrantic muscular control by; a preliminary report on result of experiments on vestibular apparatus of pigeon; Woolsey 259
experimental vestibular tremors, with analogous clinical cases; C. L. Woolsey *314

Walshe, F. M. R.: Decerebrate rigidity of Sherrington in man, its recognition and differentiation from other forms of tonic muscular contraction *1
War and acute delirious hyperthymia; Benon 466
Wassermann test, Wassermann reaction and colloidal method in spinal fluid of neurosyphilitics; theoretic considerations on Wassermann test; Rizzo 467
Weisenburg, T. H., and Stack, S. S.: Central pain from lesions of pons ... *500
Wilson, G.: Crural monoplegia and paraplegia of cortical origin with discussion of cortical centers for rectum, bladder and sexual functions. *669
Winterode, R. P., and Lewis, N. D. C.: Porencephalic defect associated with tuberculous encephalitis, histopathologic support to Strümpell theory of inflammation *304
Woolsey, C. L.: Experimental vestibular tremors, with analogous clinical cases *314
Wright, H. W.: Chronic intestinal amebiasis, clinical aspects with special reference to neuropsychiatric manifestations *226

Xanthochromia: See Cerebrospinal fluid, xanthochromia

Zona: See Herpes zoster

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CONTENTS

PAGE

NOTES ON A SERIES OF INTRACRANIAL TUMORS AND CONDITIONS SIMULATING THEM. HARVEY CUSHING, M.D., BOSTON 605

CRURAL MONOPLEGIA AND PARAPLEGIA OF CORTICAL ORIGIN WITH A DISCUSSION OF THE CORTICAL CENTERS FOR THE RECTUM, BLADDER AND SEXUAL FUNCTIONS. GEORGE WILSON, M.D., PHILADELPHIA 669

THE PSYCHIC MANIFESTATIONS IN PARALYSIS AGITANS. J. ALLEN JACKSON, M.D., G. B. M. FREE, M.D., AND HORACE VICTOR PIKE, M.D., DANVILLE, PA..... 680

NEWS AND COMMENT:

ASSOCIATION FOR RESEARCH IN NERVOUS AND MENTAL DISEASES 685

DIETS AND FASTING IN EPILEPSY..... 686

CENTRAL NEUROPSYCHIATRIC ASSOCIATION..... 687

ABSTRACTS FROM CURRENT LITERATURE..... 689

MORPHOGENESIS OF THE NERVOUS CENTERS IN CHIROPTERA.—ASTHMA AND VAGO-SYMPATHETIC IMBALANCE.—THE TREATMENT OF BRAIN ABSCESS.—TAY-SACHS' DISEASE.—PARAPLEGIA DUE TO A BULLET COMING ON FOUR YEARS AFTER THE WOUND.—ARTIFICIAL NERVE BRANCHES FOR INNERVATION OF PARALYZED MUSCLES.—GUNSHOT WOUNDS OF THE HEAD.—TRYPARSAMID IN NEUROSYPHILIS.—SKIN SIGNS OF VISCEROSENSORY PHENOMENA IN ACUTE APPENDICITIS.—EXOPHTHALMIC GOITER AND THE INVOLUNTARY NERVOUS SYSTEM.—COAGULATORY ACTION OF PITUITARY EXTRACT.—STUDIES IN EXHAUSTION: HEMORRHAGE.—ALCOHOL AND BEHAVIOR OF WHITE RATS.—THE CARDIOVASCULAR SYSTEM IN THYROID DISEASE.—EXPERIMENTAL TRAUMATIC SHOCK.—CHOKED DISKS IN EPIDEMIC ENCEPHALITIS.—RHEUMATIC FEVER SIMULATING ENCEPHALITIS LETHARGICA.—SYPHILIS AND MENTAL DEFICIENCY.—ARSENIC IN THE SPINAL FLUID AFTER ADMINISTRATION OF ARSPHENAMIN.—CALCIFICATION AND OSSIFICATION OF THE MENINGES.—TREATMENT OF MIGRAINE WITH PEPTONE.—MONGOLISM IN ONE OF TWINS.—PRESERVATION OF THE FACIAL NERVE IN THE RADICAL TREATMENT OF PAROTID TUMORS.

SOCIETY TRANSACTIONS:

NEW YORK NEUROLOGICAL SOCIETY..... 709

CHICAGO NEUROLOGICAL SOCIETY..... 715

BOOK REVIEWS 719

INDEX TO VOLUME 10..... 725